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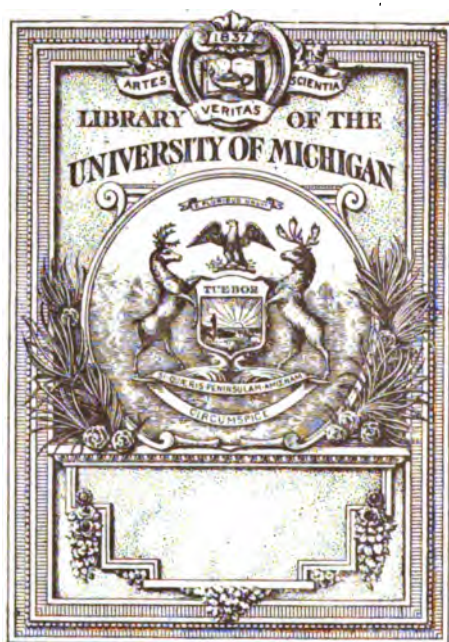
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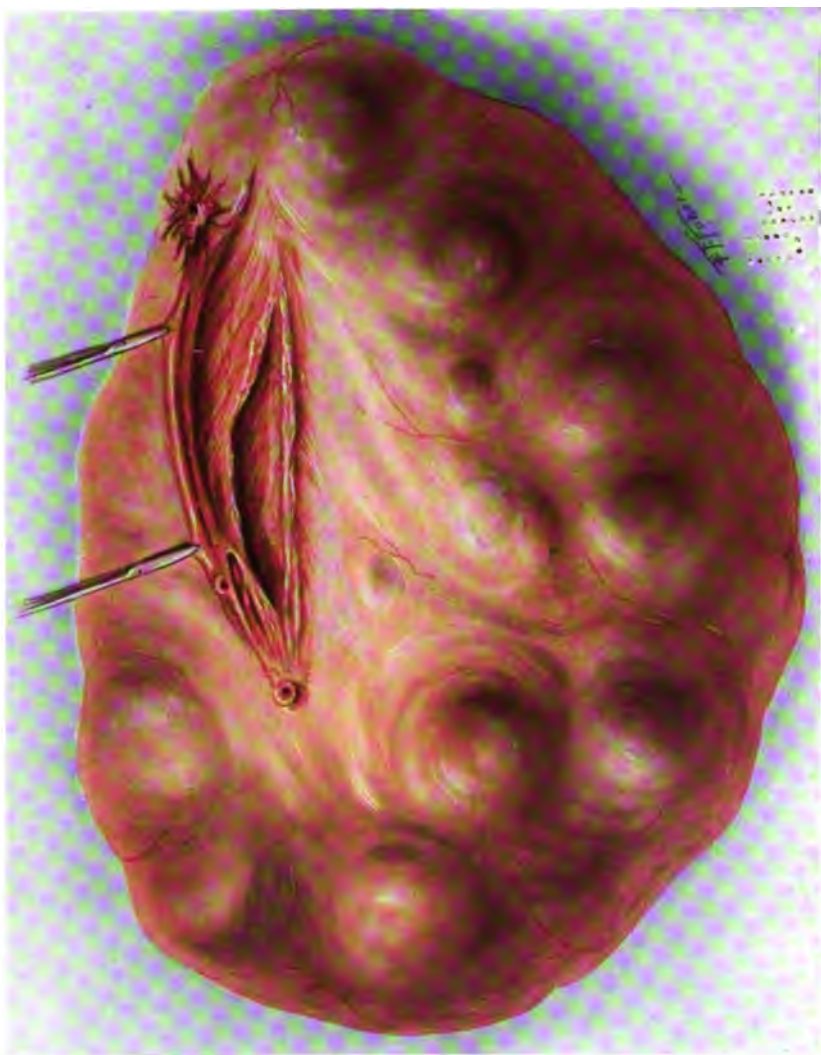
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Ovarian cystoma, weighing thirteen pounds. Removed four weeks after normal delivery. Case No. 1. *Page 62.*

# INTERNATIONAL CLINICS

## A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND  
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-  
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PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
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HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD

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# Clinics

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## THE RELATION OF NERVOUS DISEASES TO HEART AND BLOOD-VESSEL DISEASES

By **LOUIS FAUGERES BISHOP, A.M., M.D., Sc.D., F.A.C.P.,**  
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---

**GENTLEMEN:** The task that I have undertaken to accomplish in a single hour is hardly compatible with the time at my disposal, namely, the relation between the nervous system and disorders of the heart and circulation. However, the subject is so much a matter of negatives that perhaps I can cover the main points.

The relation between the nervous system and disorders of the heart and circulation is necessary to be understood, particularly from the point of view of treatment. The belief current with the laity and profession is quite erroneous. Instead of being a concrete factor in cardiovascular disease, the nervous system exerts its effect chiefly in an indirect manner. The heart and blood-vessels constitute essentially an independent system, and while there is an organic connection whereby the nerves may at times modify action, as a rule, the modification attributed to the nerves is due to chemical effects.

I have had the opportunity in past years, as chief of a division in a neurological clinic and as visiting physician to an extensive hospital service, to observe many examples of serious nervous disease. The most striking general impression has been how well the heart and blood-vessels have behaved in the presence of most serious diseases of the nervous system, providing the chemistry of the body had not first suffered from the depressing influence of the nervous system.

That even the controlling influence over the heart by the cardiac nerves, namely, the accelerators and the vagi, in health, is probably also chemical is suggested by Hirschfelder.

Suggestive of this same fact is the truth that the same drugs

which quiet the nerves do not exert a similar influence on the heart, the bromides being a most conspicuous example; nor do the same drugs which stimulate the nervous system have a similar effect on the heart, strychnine being a most notable example.

The subsequent history of many cases of supposed nervous disorder of the heart proves that in reality the heart is suffering from some toxic influence and developing organic changes. Sometimes, as in diphtheria or other infectious diseases, the particular influence is easily arrived at. In others, while the particular poison cannot be identified, the results of treatment directed against various types of food poisoning strongly indicate the underlying cause.

Cardiac neurasthenia is a name given by prominent authors to a group of cases which MacKenzie calls the "X" disease, and which I myself described some years ago as "Constitutional Low Arterial Tension." MacKenzie's description, which I will have to omit, is very interesting.

The indirect effects of depressing psychic diseases upon the heart is, of course, well known—the cold fingers and bad circulation of melancholia and so on. But I am disposed also to believe that even in these cases the effect is indirect, and is due either to a coincident chemical defect or aberration rather than the direct effect of the disease.

So my point of view and positive belief is that the circulatory effects usually attributed to nervous disease are, in fact, due to the same causes as those of the nervous disease. There is this in common between circulatory and nervous diseases: that in many cases they have the same origin.

You, no doubt, have heard a great deal during this course about so-called auto-intoxication. It is, indeed, a great subject and one that needs to be understood. My own point of view, and I have spent many months and examined many thousands of specimens with this in mind, is that the main causes of most circulatory diseases and functional nervous diseases are found in the wrong disposition of the protein food, and I find, as a matter of fact, that most so-called functional heart disease, instead of being due to the nervous system, is really due to chemical poisoning. I could cite many cases if time permitted.

I can outline in a few words my idea of the chemical relation of

food to heart disease. You know that nearly every person is idiosyncratic to some form of food—some can't eat crabs, others oysters, others eggs, milk, and so on. All along the line you find individuals who are idiosyncratic to some form of food. In these people these articles of food cause conscious disagreeable symptoms, and the person knows that these things harm him and is apt to avoid them, so they are idiosyncratic with conscious disagreeable symptoms. I believe that a large number of persons possess this idiosyncrasy to particular grouped amino-acids which come from protein food which does not produce conscious symptoms, but causes damage. This goes on for years until the heart and blood-vessels are damaged, and we have the insidious development of heart disease. If the acid is pressor instead of depressor, we have the development of high pressure and damage to the cardiovascular system.

I have had to condense so much that I will repeat if any points are not clear.

It is a very wonderful thing when we think of it that every animal and bird has a particular protein molecule, so we have an indefinite number of proteins from the different animals, which means many different combinations of amino-acids. As many as fifty different amino-acids have been recognized and studied in the laboratory, and there are probably fifty times fifty of these amino-acids.

Every animal is able to take the protein molecules of every other animal into his system and break down that molecule and reassemble it so that it conforms to the construction of his own particular kind of molecule. If a bird eats dog meat the resulting flesh is bird, and if the dog eats bird meat the resulting flesh is dog meat, and that is the crux of the whole matter.

Some of the amino-acids are poisonous and are kept out of the circulation by the liver and from these simple amino-acids the protein molecule is built up. You can make amino-acid from the human placenta. From various foods you get amino-acids that act as depressors. Some people take out of eggs amino-acids that cause most horrible migraine. I believe that most cases of chronic cardiovascular disease are examples of amino-acid toxicardia, and the heart in these cases is affected by these poisons derived from the protein food, either because there is putrefaction in the intestine, the importance of which, I think, has been tremendously exaggerated, or because the

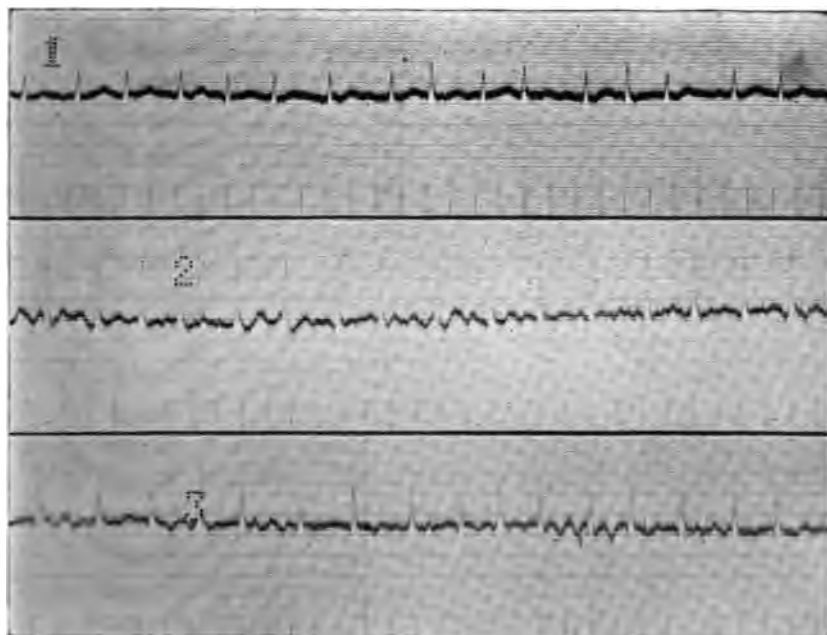
ultimate chemistry of the body has been broken down. In other words, the individual is not able to protect himself from certain amino-acids that get into his circulation. I see every day of my life such marvellous recoveries from cases of serious cardio-vascular disease in which I have reduced the number of food proteins to the lowest possible terms, thus cutting out the amino-acids to which the individual is idiosyncratic.

Out of a great many people I have asked this man to come to-day, because he illustrates very well a case of chronic protein poisoning which has done very well in two or three weeks on a regimen in which his food has been cut down to a single article of protein food. He also illustrates another important point and that is, that in a great many instances when the heart is poisoned the most delicate part suffers out of proportion to the rest of the heart. In other words, in these severe cases of amino-acid poisoning the auricle suffers more than the ventricle, due to toxic causes. His pulse when I first saw him was 144 and absolutely irregular in force and rhythm. His pulse has been brought down to 90 on digitalis. He has taken large doses of castor oil and has lived on a diet of bread, butter, vegetables, fruit and tomatoes, and has derived his protein from a single article of food, cheese. Cheese is the safest single protein food we know of. So I cut out all amino-acids except what we find in cheese. You see, he looks like a pretty sick man yet, but when I saw him first he looked a great deal sicker, but I have every expectation that he will recover and be able to take up his regular life, though he may not recover from his fibrillation of the auricle. This man had been all over Germany and had taken baths and so on, but with no effect, and yet he has yielded to treatment based on this theory very quickly.

Now, if the causes of irregularities of the heart have very little to do with the nervous system, and such is my belief and the belief of most workers in cardiology at the present time, what is the mechanism of cardiac irregularity?

No matter how carefully the heart is isolated from all nervous controlling influence, it continues to beat in a rhythmic manner. Of course, as you know, we believe that the nerves have very little to do with it, and that the so-called muscle theory as opposed to the nerve theory is not a true explanation.

**FIG. 1.**

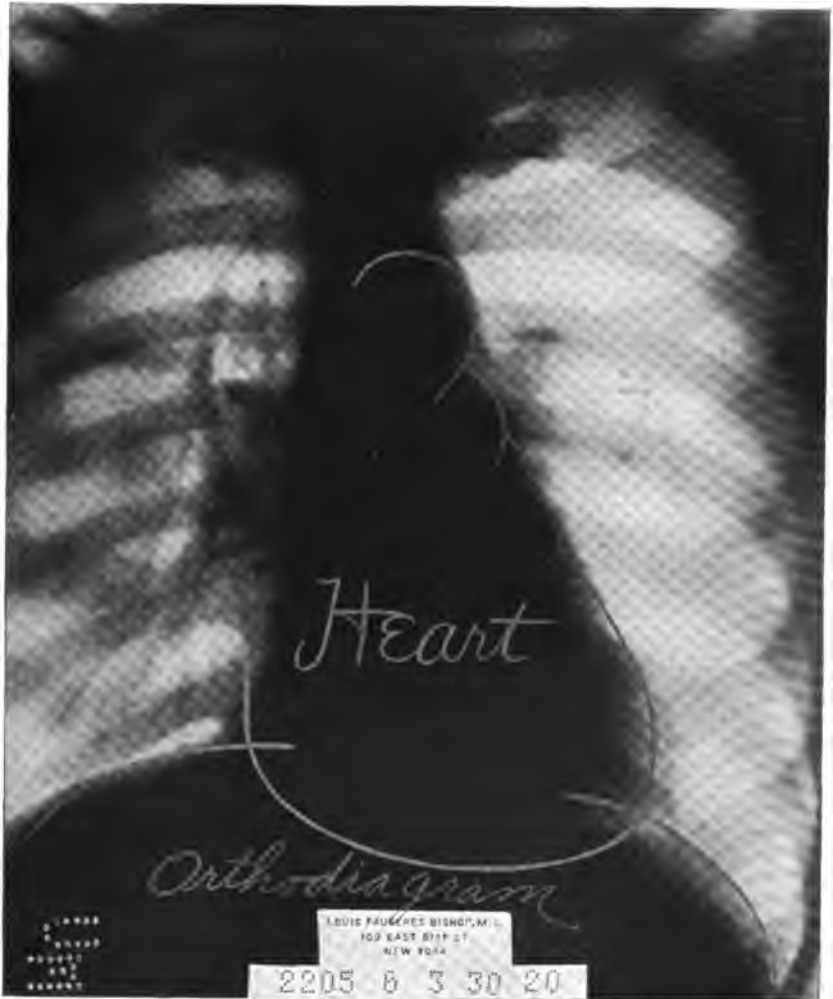


**Fibrillation of the auricle.**



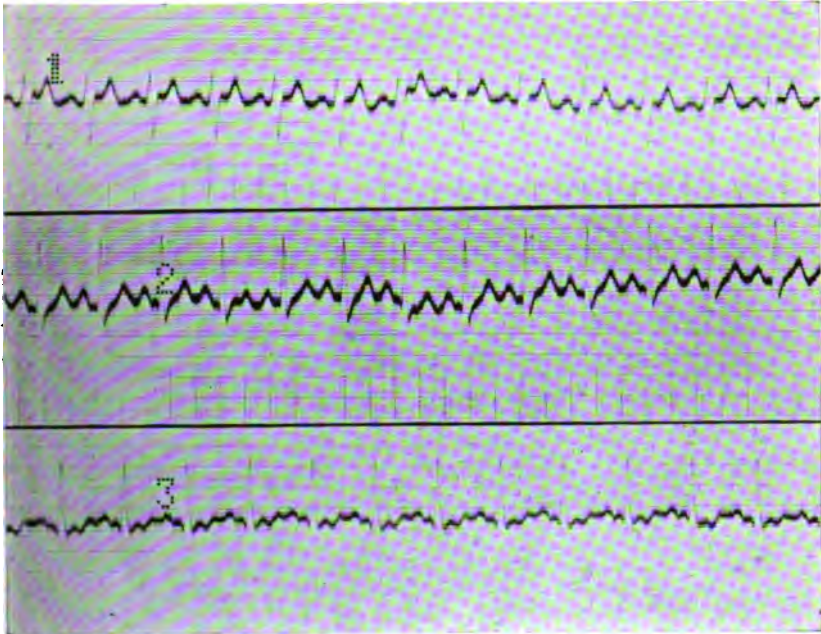


FIG. 2.



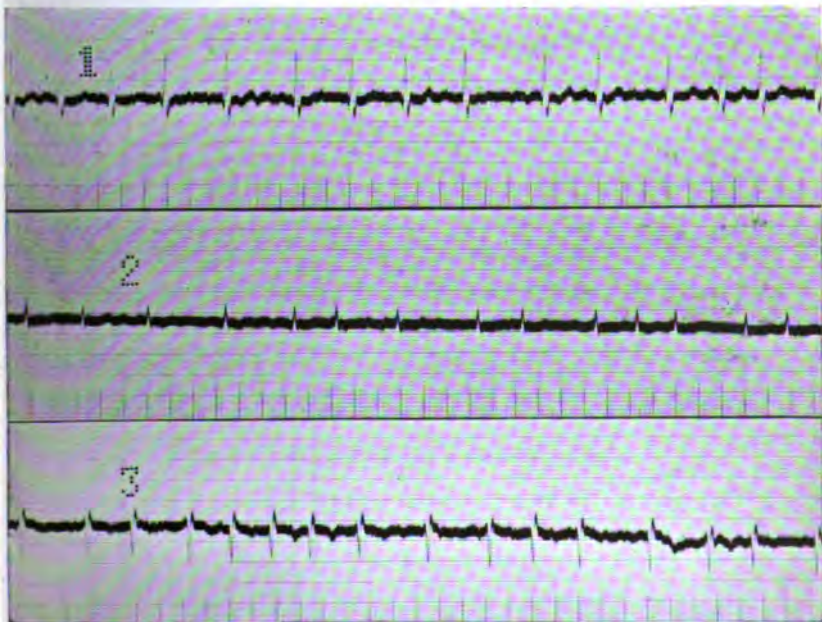
Orthodiagram of heart.

FIG. 3.



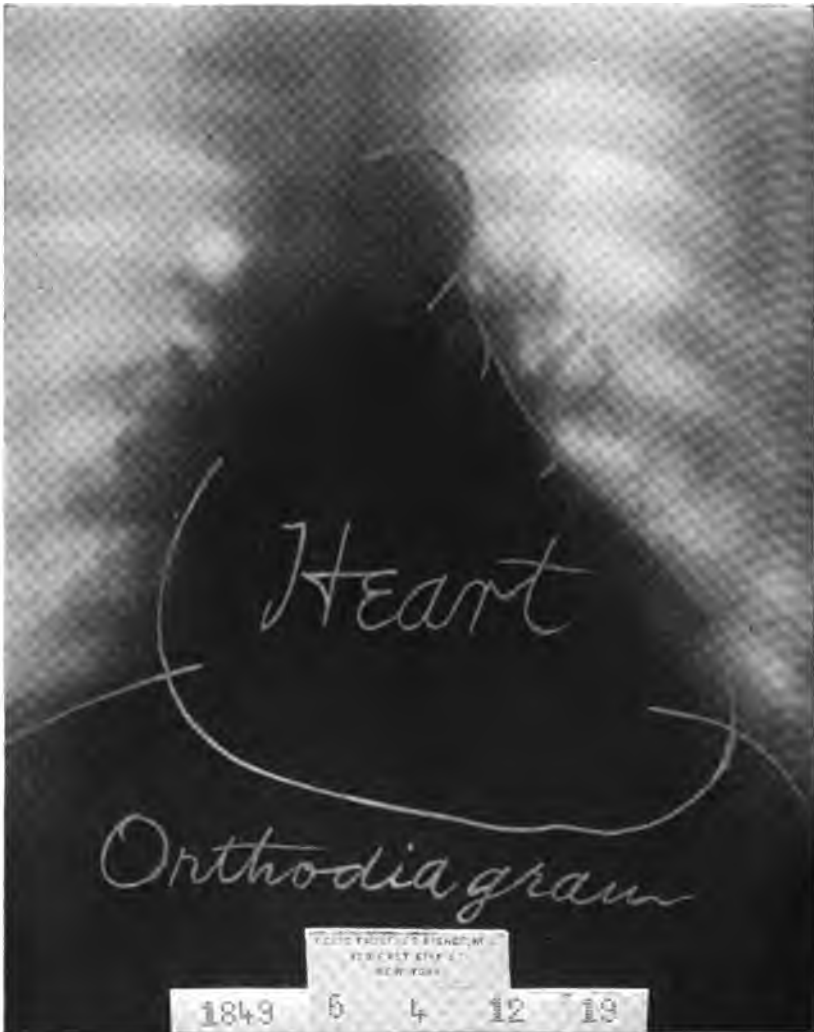
Rapid heart in Grave's disease.

FIG. 4.



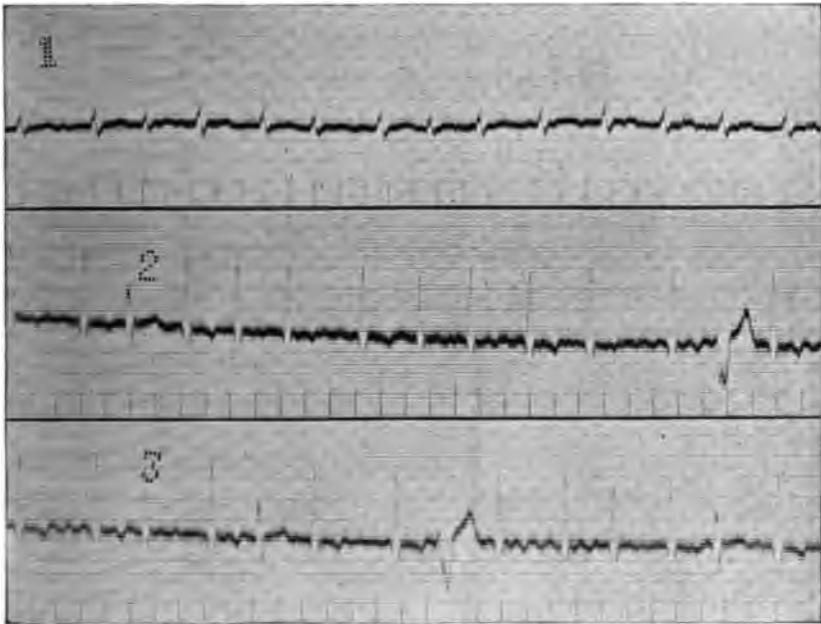
Fibrillation of the auricle.

FIG. 5.



Orthodiagram from a case of auricular fibrillation.

**FIG. 6.**



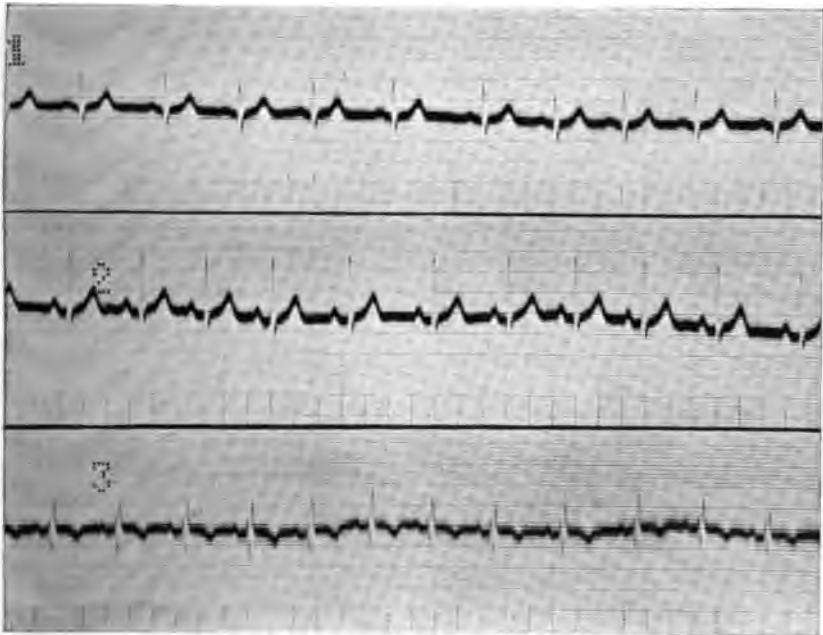
**Fibrillation with ventricular extra systoles.**

FIG. 7.



Orthodiagram from a case of fibrillation.

FIG. 8.



Sinus arrhythmia.

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I want to show you (I hope I am not going into things that are too simple) the various forms of cardiac irregularity and the means of recognizing them, disclaiming any particular influence of the nervous system.

I do not believe any longer in tachycardia of nervous origin, because in nearly every instance we are able to prove some other cause. Nobody has ever been able to prove or work out the relation of the sympathetic nerve to the heart, and the vagus action is very indefinite except in some cases where the vagus can be stimulated; and the accelerators seem to act very little because the heart acts just as well when separated from it as not.

I meant to have a man here to-day whose ventricle is absolutely cut off from his auricle, yet that man's heart beats regularly 28 a minute—more regularly than it would if it were under nervous tension. This case has been under my observation for nine months, and the pulse never varies from 28.

I will show you some of the curves taken at random in my office. Every patient has an electrocardiogram taken which is then put on record.

Here is an electrocardiogram (Fig. 1) from a patient with fibrillation of the auricle; also an orthodiagram (Fig. 2). You see he had the same condition as this man exactly. This man was an actor and came under my care with a pulse of 144. On digitalis and a proper diet he was able to carry on his work and is continuing it to this day as far as I know. This is his tracing, and you see in the tracing the entire absence of the characteristic auricular current just as this man shows complete absence of the auricular event. You see the pulse is completely irregular and that in these are irregularities between the beats representing the fibrillation of the auricle.

Here is a case of Grave's disease in which the heart is (Fig. 3) simply very, very rapid, but it is very interesting because you notice that it was beating more than twice a second—it was beating 120.

Another example of fibrillation of the auricle (Figs. 4 and 5). Figs. 6 and 7 are examples of fibrillation with ventricular extrasystoles. I want to say just a few words in conclusion to recapitulate what I have said. I think the relation between the nervous system and disorders of the heart and circulation is indirect and is only the relation of conditions due to a common cause. I think the cause in



nearly all cases is chemical. This man illustrates very well a case of toxicardia. He did not do very well until he was cut off from all amino-acids, except those found in cheese. After putting him on a diet of that kind and castor oil once a week, his general condition has improved to a considerable extent.

Fig. 8 is an example of sinus arrhythmia.

That is the particular lesson I would like to bring to you. I am either very wrong about this subject or I am very right, and if I am right, it is very important. A great many people with cardiovascular disease are suffering from poisoning of the heart muscle. I do not like the word auto-intoxication because it is so mixed up with intestinal putrefaction and does not work out in practice, but a diet of few proteins and outdoor exercise does work out in practice, and these serious cases of cardiovascular disease do well on that kind of treatment. I think a good many of the nervous cases would do well on the same kind of treatment. I am not much of a believer in the bacteriology of the intestinal tract (Fig. 7), as the key to the etiological factor in cardiac conditions and nervous diseases.

## A CASE OF CHYLOTHORAX

WITH AN ANALYSIS OF THE PLEURAL FLUID BY PROFESSOR WILLIAM H. WELKER

By CHARLES SPENCER WILLIAMSON, M.S., M.D.

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THE patient before you is a native of the United States, of Irish descent, aged forty-three years, by occupation a painter. He came into the hospital yesterday morning at 7 o'clock, being brought in on a stretcher. On entrance he complained of two things: First, extreme shortness of breath; second, swelling of the body. I may say that much of the history has been elicited from relatives and in imperfect snatches from the patient himself in his lucid intervals, for he has been very rambling and incoherent. From various sources, then, we can describe the *onset* and *course* as follows:

Six days ago the patient had a smothering sensation in the chest after walking downstairs. This lasted for about fifteen minutes and then passed off. He says he continued to feel well up to yesterday afternoon, when he attempted to lift a plank. He then developed this smothering sensation again, only this time to a very much greater degree. He became very short of breath and has been in that condition since. He states that his body began to swell up only yesterday. He vomited a little yesterday and again to-day. I have done my best to check up this story from the relatives, and they all insist that he has been able to work, although they think that he has not been quite so well recently. The best proof that he was working is that he was removing a plank from the ladders when he developed the last smothering attack.

*Symptoms in Detail.*—He assures me that the shortness of breath was never present before the onset of this disease. Since that time he has noticed that his breathing was very fast, and a nurse who counted it said he was breathing thirty-five to forty times per minute. The swelling of the body, both he and his family insist, has come on since yesterday. The patient states that he has never had even the least swelling of the legs before. We will discuss that statement later and see if it seems probable.

*Previous Illnesses.*—He has had occasional colds and coughs besides the usual diseases of childhood. Although he had an occasional sore throat, he had never had any rheumatism. In the last six months he has lost twenty pounds in weight.

*Habits.*—He is accustomed to drinking a pint of beer with each meal and several drinks of whisky a day. His bowels are regular, urination is normal. He chews tobacco to excess and smokes occasionally. His sleep and appetite have been good up to the last week. All venereal infections are denied.

*Occupational History.*—He is a painter of houses, and in this work he has been using lead paints constantly for the past ten years.

*Family History.*—His mother died of some sort of tumor at the age of seventy-two; his father died of old age at eighty-three. The patient was married for fourteen years, his wife dying in the last child-birth. His wife had four miscarriages and five full-term children; the children, however, all died either at birth or under one year of age. The patient's condition is now substantially the same as it was when he came into the hospital, so I will go over his physical examination with you.

*Physical Examination.*—As you see, the patient is a man of apparently forty-eight or fifty years of age, exceedingly dyspnoic, and when we turn down the bed-clothes we see that he has a very marked generalized œdema, that of the ankles being quite pronounced and rather soft. This point is important, in my judgment, because a soft œdema means a relative recent œdema. An œdema which has lasted for a long time becomes more cellular, the so-called "solid œdema."

*Head and Neck.*—The scalp is negative. The pupils are moderately dilated, equal, and react promptly to light and accommodation. The teeth and gums are in fairly good condition and there is no evidence of a lead line. When I looked at his gums carefully with a hand lens, I could see none of the bluish, punctate appearance which is so characteristic of lead intoxication. His throat is negative. You will notice that he prefers to lie on his left side and you see that there is marked œdema of the left side of the face. This, I take it, means that he has been lying on the left side to the practical exclusion of the right. The color of his face is a peculiar lavender tint, a combination of flushing and cyanosis. The veins of the neck stand out promi-

nently, but there is no especial pulsation in them except that communicated from the carotids.

*The Chest.*—The chest wall is œdematous on the left side; the respiratory excursion is practically gone on the left side and somewhat impaired on the right. Palpation discloses the same thing. On percussion the findings are striking. The entire left side of the chest is absolutely flat, the flat note extending from the second rib in front all the way down (Fig. 1). It is flat also for fully one inch to the right of the sternum, and behind the flatness extends over the entire chest except for a very small place above the spine of the scapula. Over this flat area the tactile fremitus is entirely absent and the vocal fremitus practically so. A very little bit can be obtained over the upper portion of the lung. Distant bronchial breathing can be heard over the left apex with a few râles, but below this no respiratory sounds can be heard. The right side of the chest wall is not œdematous. The tactile fremitus is substantially normal over the right lung, and on percussion the note is slightly skodaic. On auscultation (Fig. 2) the breath sounds are slightly harsh over the entire lung, and a good many small, moist râles can be heard, especially posteriorly at the base.

*The Heart.*—The apex-beat cannot be accurately located. More properly speaking, we can find a pulsation over to the right of the sternum in the fifth intercostal space about two fingers to the right of the sternal edge, but we are not at all sure that this actually represents the apex of the heart. On the contrary, I suspect strongly that it does not. There is a diffuse and very weak pulsation behind the lower portion of the sternum, especially in the right half. The flatness of the heart merges directly both above and to the left into the flatness already described. On auscultation the tones are feeble, the rate is quite rapid, it now being about 110. No thrills can be felt and no friction-rubs.

*The Abdomen.*—The abdominal wall is œdematous, particularly on the left side. Yesterday we thought that we obtained a distinct fluid wave with some shifting dulness, but to-day I am very doubtful about it. At any rate, we can say with certainty that there is no considerable amount of fluid in the abdominal cavity if, indeed, there is any. The spleen is not palpable; the edge of the liver can be indistinctly felt about two fingers below the xiphoid cartilage. The

genitalia are negative except for a small scrotal hernia. The patient also has a small left inguinal hernia which has been there for about two years.

*The Extremities.*—As we have already noted, the legs are somewhat œdematous, especially the left, but the left arm and hand are only slightly so, and the right arm and hand are not œdematous at all. The skin is pigmented and shows a great many scratch marks on the body, undoubtedly due to an old pediculosis.

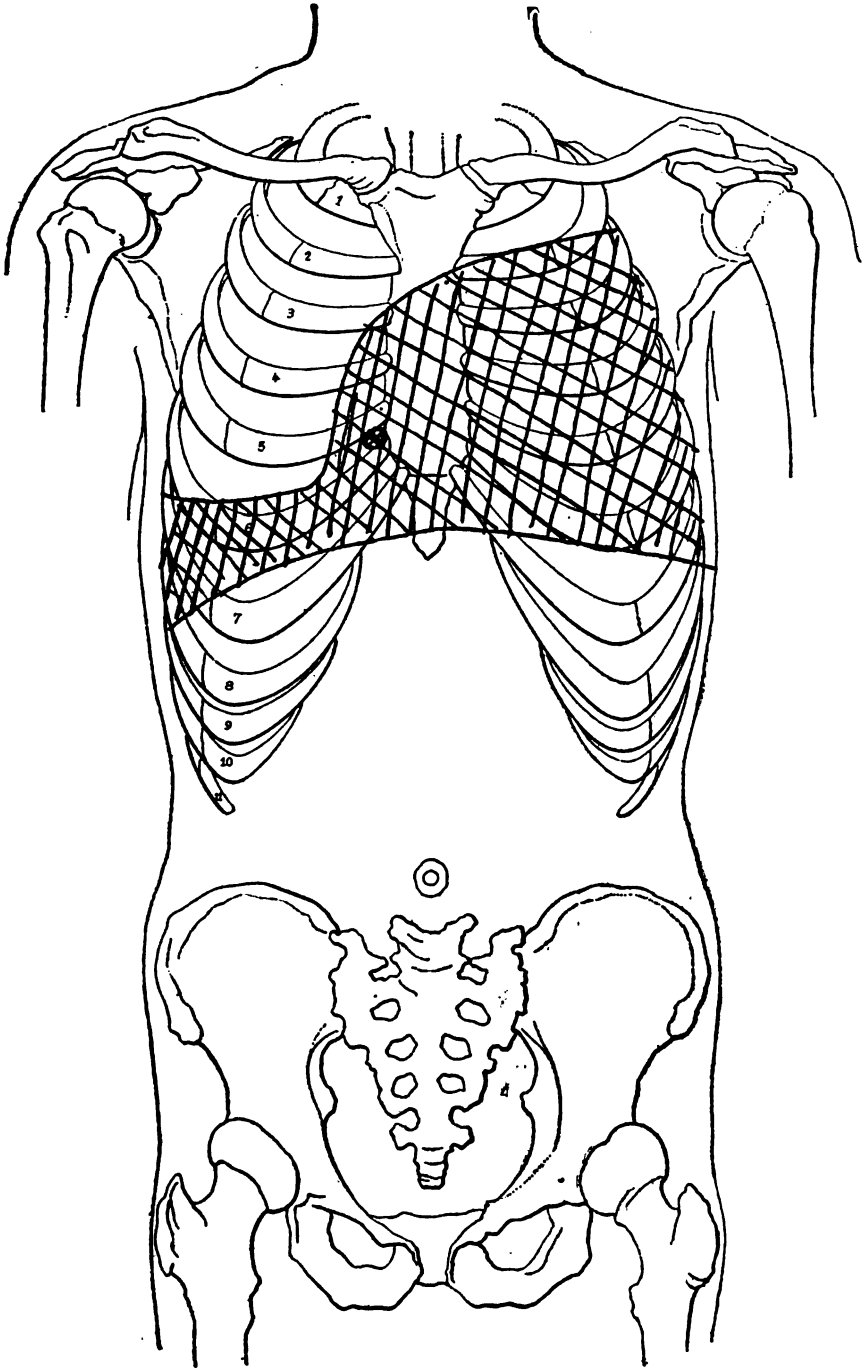
*The Reflexes.*—The patellar reflexes are diminished but present. The rest of the reflexes are substantially normal.

Our general impression of this patient is that he is desperately ill; indeed, so much so that I hesitated to bring him into the clinic, but because of the great interest attaching to the case I decided to do so. Mentally he is much more lethargic than when he came in yesterday and seems very much sicker. The pulse is certainly much weaker now than it was then. The blood-pressure was 132 yesterday, to-day it is 115. The urinalysis is incomplete, inasmuch as we have not been able to secure a twenty-four-hour specimen, but the single specimen examined shows an acid reaction with a trace of albumin and a great many fine granular casts and an occasional leucocyte. The blood count shows 9100 leucocytes, and a differential count gave 71 per cent. polymorphonuclears, 26 per cent. small mononuclears, 2 per cent. large mononuclears, 1 per cent. basophils.

*Diagnosis.*—When we analyze this case we see that the history is not very satisfactory. So far as it can be obtained from the patient and from his sister, who lives with him, he seems to have been taken sick only about a week ago. This, however, is a little difficult to reconcile with the very manifest cardiac insufficiency which he shows, and, in particular, with this enormous area of flatness in the left chest. I think it is hardly worth while for me to go into the differential diagnosis of this flatness, because it seems perfectly clear that there is fluid there, and my interne made the diagnosis of an enormous pleuritic effusion within a few minutes of the time when the patient entered the hospital. It is difficult to understand how a patient could be working or, indeed, existing with this enormous effusion, which seems to extend right up to the clavicle and which has pushed the heart away over to the right.

I happened to be in the hospital a few hours after the patient entered, and my interne had just finished making an exploratory

FIG. 1.

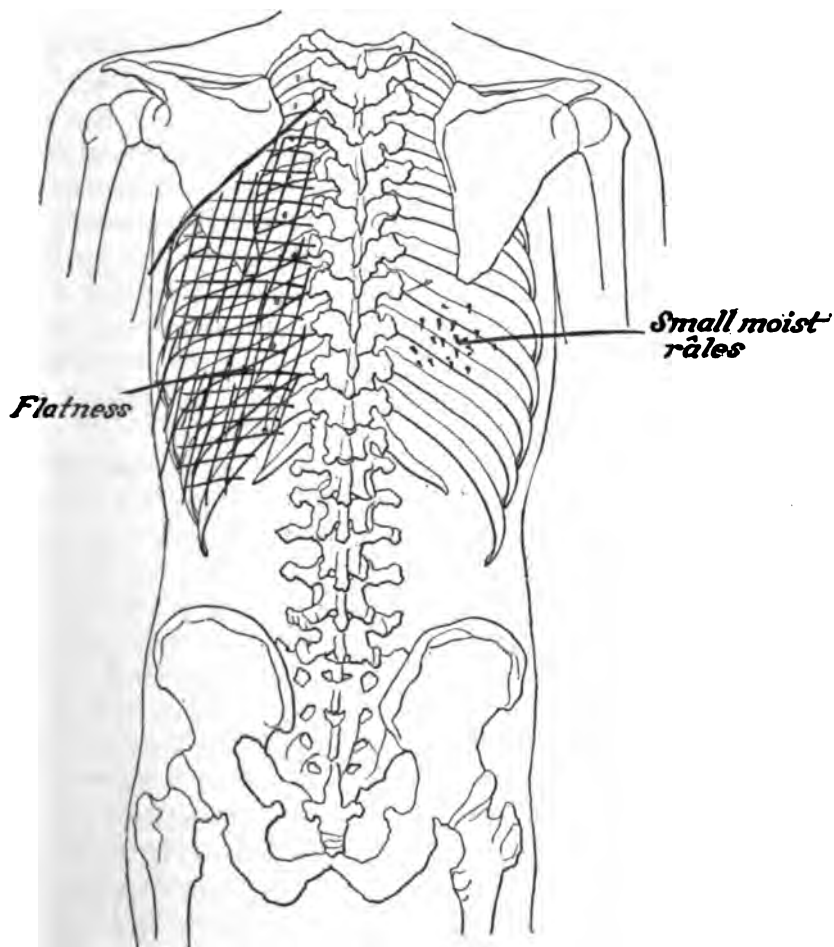


Chylothorax, anterior view. Flatness over shaded area.

puncture, and this is where the particular interest in this case centres. To our intense surprise the fluid withdrawn instead of being serous or possibly hemorrhagic, as we expected, was of almost exactly the appearance of a moderately rich cream; that is, of a whitish-yellow color, rather thick consistency, and having an odor which is best described as somewhat oily. The specific gravity was 1020, and the examination of this fluid microscopically showed rather striking findings. Not a cell was to be seen except an occasional erythrocyte, which was unquestionably due to the puncture wound. After removal of the albumin by acidulation with acetic acid and boiling, no sugar could be determined. On staining, no bacteria could be found, and a culture made just twenty-four hours ago has remained sterile up to the present. The other things which could be seen under the microscope were a moderate number of droplets and a very large number of very characteristic crystals. I need not describe these to you any further than to say that they are of the peculiar shape which is so characteristic of cholesterol. In a nutshell, we found nothing but these small droplets, which are presumably fat, and a very large amount of cholesterol, which is the striking feature under the microscope. Only 50 c.c. were removed at this exploratory puncture. As the fluid came through the syringe my interne's first impression was that it was pus, but a little closer inspection showed that this was not the case, but that he had before him one of the extremely rare cases of chylothorax. Later in the afternoon another aspiration was made for the purpose of removing a sufficient quantity of the fluid to alleviate the pressure. The aspiration was very simple and everything went smoothly, the fluid being removed very slowly, until a little over 1 litre had been removed, when the patient developed the most alarming sensations—coughing violently, becoming very restless, the pulse becoming very weak, almost imperceptible, and the aspiration had to be interrupted. The fluid removed was the same as the first, a creamy white fluid, which I show you here in this flask (exhibiting bottle), and I think I might hand it around the class as a specimen of cream and none of you would suspect the imposition. It has almost the exact appearance of cream, not being merely milky, but like good, honest cream.

Of course, the diagnosis is plain. It is practically impossible to conceive of any other diagnosis than that of chylothorax, using

FIG. 2.



Chyllothorax. Posterior view.



this term purely in a descriptive sense, and not implying that it must necessarily be due to an actual lesion of the thoracic duct. The next point which I want to impress upon you is the extreme rarity of these cases. This is the first case which I have ever seen, although I have had an opportunity of seeing several cases of chylous ascites, in none of which, however, was the fluid more than faintly milky. I have taken occasion to go through the literature as well as I could since yesterday, and find that up to 1895 Bargeduhr had only been able to collect forty cases of this type. He estimates that cases have been appearing at the rate of about one in six years. Now, inasmuch as the diagnosis can readily be made by aspiration, and inasmuch as the findings are so perfectly characteristic that it is practically impossible to mistake a chylous fluid for anything else, it is not likely that there have been many cases overlooked, and I suppose it is a fair statement to say that the total number of recorded cases is still quite small. The great rarity of the cases and the very striking features shown in this individual leads me to make a few further remarks.

*General Considerations.*—In a general way we may use the current classification of chylous and chyloform effusions. By “chylous effusion” is meant one in which the appearance is due to an admixture of chyle. The chyloform effusions are those which simulate the chylous effusions in appearance, but are not due to admixture of chyle. There is a good deal of uncertainty about these cases. In most instances chyloform cases are due to fatty degeneration of the cells in the exudate, or perhaps in some instances the chylous appearance is not due to fat at all, but to other substances, such as lecithin or various albuminoid substances. In other cases it may be due, at least in part, to cholesterol. The cases of chylothorax are generally due to things which interfere with the integrity of the thoracic duct at some part of its course. I have copied here from the work of Bargeduhr the etiology in the cases collected by him and will read them to you.

In eleven cases the cause was uncertain or not determined. In twenty-two cases it attacked the right side nine times, the left four, and both sides five. In four cases the side was not stated; thirteen of the cases occurred in males, eight in females, and in one the sex was not given.

## CHYLOTHORAX

	Cases
Gunshot wound .....	1
Compression of thorax by tumor .....	1
External force .....	4
Parasites .....	1
Thrombosis of the left subclavian vein .....	3
Effusion, non-tuberculous .....	1
Disease of lymphatic vessels .....	1
Carcinoma .....	4
Overexertion .....	1
Stoppage of the thoracic duct .....	1
Malignant lymphoma .....	1

## HYDROTHORAX CHYLIFORMIS

	Cases
Pleuritic effusion, simple .....	3
Pleuritic effusion, tuberculous .....	1
Abscess of the lung .....	1
Carcinoma of the pleura, lymph-vessels, and lymph-glands..	4

In regard to the etiology in the case before us, we can be sure only of one thing, and that is that the patient has an enormous exudate in his left chest and this is manifestly not a part of a general anasarca. It is quite doubtful whether there is any fluid in the peritoneal cavity or not. Yesterday I thought there was a small quantity, but to-day I am unable to demonstrate it. As the bowels have been thoroughly cleared out since, it is not improbable that he had a small amount of fluid in the colon. If, now, this fluid is not a simple hydrothorax it is evidently a pleuritic exudate of some sort. It seems to me exceedingly unlikely that such an enormous quantity of fluid under such high pressure could have accumulated in a week's time. The low leucocyte count makes us think very strongly that this is probably a tuberculous exudate, since a simple—that is, a non-tuberculous exudate—would probably have shown a marked leucocytosis, and we would not have found the fluid sterile. I may say, however, that we have examined the fluid very carefully for tubercle bacilli by the Löffler method, with negative results. This, of course, does not justify us in saying that the fluid is not tuberculous, since, as you very well know, it is sometimes very difficult to find tubercle bacilli in a fluid obtained in this way. The patient's temperature is practically normal, 99° F. being the highest point reached up to date.

Again we must ask ourselves is it possible that this cardiac breakdown is only a week old? It certainly seems fresh, and yet my judgment is that it is older than that. I doubt if we will be able to obtain any more light on the subject unless we succeed in emptying the pleura of its contents, and after our experience with the aspiration yesterday we shall certainly have to proceed very cautiously. At present the patient seems so sick as to render his recovery doubtful. We might think of the possibility of his having a tuberculous effusion which has opened, by ulceration, the thoracic duct, so that there has been an effusion of chyle into the chest. The fluid is being analyzed chemically, but I have not had the report as yet in regard to its composition. If it is a true chylous effusion, it is certainly not very recent.

On the other hand, there is another possibility which is to be reckoned with, namely, that this effusion is a very old one. Every physician has seen examples of tuberculous exudates in the chest which have been unrecognized for a long time, and which have come on so gradually that the patient has considered himself in good health up to a short time before consulting the physician. I cannot help feeling that that is probably the case in this patient. In spite of his own and his sister's assertion that he was working up to a week ago, or even since then, I feel that the smothering sensation a week ago indicated that the effusion then had attained quite stately proportions. I do not know of any way to settle this question definitely except in case of the patient's death. A necropsy, if we can obtain one, might settle the matter. It is conceivable, of course, but improbable, that he has a carcinoma of the lung or of the mediastinum. We attempted to have an X-ray taken, but he was in such bad condition that they sent him back from the X-ray department with the statement that he was too ill to have this done satisfactorily. The number of miscarriages and still-births which his wife had led us to suspect that he might be luetic, but the Wassermann report which has just come down is negative.

There is one point which, although not very decisive, must be accorded some value, and that is, that after the removal of 1000 c.c. of fluid yesterday there was absolutely no change in the position of the heart. Now this might either be that the amount of fluid in the chest is so great that the removal of 1000 c.c. makes very little

difference, or it might be that the heart has been fixed in this position. I regret that I am unable to make an accurate diagnosis of the case for you. We shall attempt to aspirate him again if his condition permits of it, and I will report to you the results at the next clinic. By that time I presume we shall have the results of the chemical analysis of the fluid, which may throw some light on it.

I wish to report the further course of the case of chylothorax which I showed you at the last clinic. I am sorry to say that it was very brief. The man died four or five hours after leaving the clinic. I moved heaven and earth in an attempt to get a necropsy, but was unsuccessful, so I am not able to add anything more in the way of diagnostic considerations to what I then said. I have, however, an analysis of the pleural fluid which I owe to the courtesy of my colleague, Prof. William H. Welker, and will put it on the blackboard for you:

## ANALYSIS OF PLEURAL FLUID

Specific gravity .....	1.0199
Solids (total) .....	6.64 per cent.
Ash (ignition at 750° C.).....	0.85 per cent.
Nitrogen (total) .....	0.75 per cent.
Nitrogen (non-colloidal) .....	0.02 per cent.
Nitrogen (colloidal, calculated as protein) .....	4.56 per cent.
Lipins (total) .....	0.79 per cent.
Lipins (unsaponifiable) .....	0.76 per cent.
Chlorin (calculated as NaCl) .....	0.73 per cent.

You will see that the total lipins are 0.79 per cent., and of those the unsaponifiable lipins are 0.76 per cent. In other words, practically all of the fat is unsaponifiable. The analysis was carried sufficiently far to determine that considerably more than 50 per cent. of this was cholesterol. The remainder was not determined. This analysis makes it, to my mind, perfectly clear that we have not to do with a fresh chylothorax. It is quite inconceivable, it seems to me, that such a fluid could have come from a chylothorax only a week or two old. Cholesterol might possibly and, indeed, often does occur in chylous effusions, but practically has always been found in combination with some fat. It seems possible, in an older case, to have

the fat completely absorbed and cholesterol deposited, but a fresh chylothorax would show much fat, which, of course, the analysis would reveal. A hasty glance through the analyses as quoted by Bargeduhr shows cholesterol in various amounts. For instance, in his Case 24, 0.34 per cent. fat, 0.38 per cent. cholesterol; and in Case 30, 0.1714 per cent. fat and lecithin, and 0.2599 per cent. cholesterol. In other words, we can assume, I think, that in the older cases the fat content may disappear entirely and the chylous appearance be due either to cholesterol alone or, more probably, to a mixture of cholesterol with protein substances.

Turning for a moment to the other angle of the case, we might ask ourselves: Was this a case of chyloform hydrothorax? It seems very unlikely, in view of the fact that the fluid is sterile, that no detritus can be seen, and no cells, that this chylous appearance should be due entirely to degenerated cells. It has been commonly held that the presence of sugar was characteristic of a true chylous exudate, but this has been shown to be inaccurate since many exudates other than true chylous ones may show sugar, and, on the other hand, some of the true chylous effusions show none, as can very well be seen from Bargeduhr's table. We examined our fluid with great care for the presence of sugar after removal of the albumin and none could be detected.

I will again show you a portion of the fluid which was removed eight days ago. It has been standing in an open vessel in the ward, and you will see that there is absolutely no trace of bad odor to it and it has not changed its appearance in the least. This rather remarkable resistance to decomposition is quite characteristic of chylous effusions.

Summing up the case we see that the great preponderance of evidence is in favor of our case being one of true chylothorax. While the clinical aspect does not admit of a decision being reached in this respect, the analysis of the fluid speaks strongly in this sense. The creamy white color, the presence of fine fat globules with total absence of cellular elements, are in themselves highly suggestive, since the pseudo-chylous fluids are milky white, the granules present do not give the fat reactions and cellular elements are often abundant. In true chylous fluids the specific gravity generally exceeds 1.012,

and the total solids are over 4 per cent. with a high fat content varying from 0.4 per cent. to 4 per cent., while in the pseudo-chylous the total solids are generally less than 2 per cent., and the specific gravity less than 1.012, and the fat content is generally low, often only traces.

I cannot refrain from referring you to the very excellent work which has been done on these fluids by Wallis and Schölberg, *Quarterly Journal of Medicine*, 1910 and 1911, volume iv. I will put their summary of the different points on the board.

#### CHYLOUS ASCITES

1. Tends to accumulate very rapidly, and in consequence large volumes are removed at paracentesis.
2. Generally yellowish-white in color and less perfectly emulsified.
3. Degree of opalescence more or less constant at successive tapplings.
4. Possesses an odor corresponding to the odor of the food ingested.
5. Microscopically the food contains fine fat globules but very few cellular elements.
6. Generally shows a distinct creamy layer on standing.
7. Specific gravity generally exceeds 1.012.
8. Depression of freezing-point about  $-0.51^{\circ}$  C., and approximating that for chyle.
9. Total solids vary considerably, but usually greater than 4 per cent.

#### PSEUDO-CHYLOUS ASCITES

1. Collects more slowly, the volume of the fluid varying with the exciting pathological condition.
2. In color a pure milky white solution in the form of an almost perfect emulsion.
3. The opacity generally increases or diminishes at successive tapplings.
4. Odorless.
5. Microscopically the quantity of free fat is variable; often numerous fine, highly refractile granules are present, and these do not give the reactions for fat. Cellular elements may be numerous and often contain fat; sometimes very scanty.
6. A cream may or may not form, but does not affect the opalescence; a sediment frequently settles out.
7. Specific gravity less than 1.012.
8. Depression of freezing-point ranges from  $-0.56^{\circ}$  to  $-0.61^{\circ}$ , and thus corresponds to the figures for blood serum.

9. Total solids rarely exceed 2 per cent.

- |  |  |
|--|--|
| 10. The total protein content generally exceeds 3 grammes per cent., and of this the serum-albumin is the largest fraction; globulin occurring only in traces. | 10. The protein constituents vary between 1 and 3 grammes per cent., and of these the serum-globulin occurs in appreciable quantities.                   |
| 11. Mucinoid substances absent.  | 11. Mucinoid sometimes present.  |
| 12. The fat content is generally high, varying from 0.4 to 4 per cent. The fat corresponds in all its properties to the fat contained in food.                 | 12. The fat content is generally low, and may be present in traces only; in its melting-point and chemical composition it proves to be pathological fat. |
| 13. Of the lipines, cholesterin is invariably found, and lecithin only occurs in traces.   | 13. The most characteristic lipine is lecithin, though cholesterin is occasionally present.  |
| 14. No evidence of the presence of a lipine-globulin combination has been given by previous observers.   | 14. The lecithin is mainly combined with the globulin, and when present is the cause of the opalescence of the fluid. Such fluids resist putrefaction.   |
| 15. The salts and the organic substances present approximate the values found for chyle obtained from the thoracic duct.                                       | 15. The salts and organic materials correspond closely to lymph and serous fluids.   |

In conclusion, it seems to me that my original tentative diagnosis of a tuberculosis of the pleura is the most likely one, although it is exceedingly difficult to exclude malignancy. The absence of blood, even in traces, and of any characteristic cellular elements in the fluid speak against carcinoma. Finally, while it would seem impossible to mistake a fluid of this nature for anything else, I must call your attention to the fact that a quick glance at this fluid might lead the careless observer to mistake it for pus, and I imagine this mistake has been repeatedly made.

## TENDON REPAIR IN THE HAND

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WE hear much these days about operations for removal of brain tumors, reconstruction of the œsophagus, extensive lung resections, while the less spectacular but more numerous and equally important minor cases are apt to be overlooked. For this reason I have chosen this patient to demonstrate to-day. He is a young chemistry student, who, while working in the laboratory about three weeks ago, cut the index finger of his right hand with a piece of glass tubing. The tubing pierced the finger on the radial side at the middle of the first phalanx, passed inwards, slightly distalwards and slightly palmwards. How deeply the glass penetrated the patient does not know. He immediately pulled out the tubing and noted that no glass had been broken off. The finger bled but slightly, so he tied a clean handkerchief around it and kept on working. That evening he went to his family physician. He had noticed that he could not flex the phalango-phalangeal joints of the injured finger. He could bend them passively, although bending the first joint seemed to pull on the wound and caused him pain. He had active control, however, of the metacarpophalangeal joint, bending the rigid finger freely upon the palm (Fig. 1). His physician painted the wound with iodine, told him to soak the finger in hot water if it pained and assured him that inasmuch as there was flexion in the metacarpal-phalangeal joint, the tendons had not been cut.

When I saw the patient about a week later the condition was as follows: The wound was slightly reddened. There was no limitation of passive motion at any of the joints of the index finger. The patient could flex the extended finger on the palm, but he could not flex either of the phalangeal joints. If the finger were flexed passively it straightened out again as soon as released.

The diagnosis was apparent. He had cut both his deep and superficial flexor tendons. These insert in the terminal and middle phalanx respectively, and when cut quite naturally the



patient is unable to crook his finger. The interosseous and the lumbrical muscles evidently were spared. These muscles inserting on the first phalanx and the extensor tendon on the dorsum of this phalanx, flex the first phalanx and extend the second and third, or, in short, bring about just the action which the patient could perform. The first physician had forgotten for the time being these little but important muscles and had presumed that the long flexor tendons were essential to any and all digital flexion.

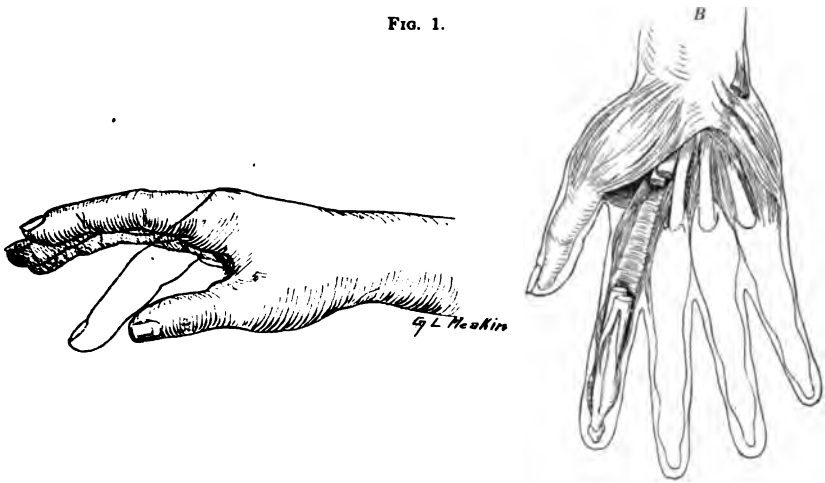
I advised the boy to wait until the wound had entirely healed and then to report to me for tendon suture. Had I seen the case immediately after injury I probably would have cleaned the wound carefully and performed a tendon repair—with catgut—but inasmuch as a week had already elapsed, I preferred waiting until every sign of infection should have disappeared.

The wound is now entirely healed, the range of motion is as I have indicated. I am going to repair the severed tendons.

While the interne is preparing the hand with tincture of green soap and water, alcohol, ether and iodine, and applying a constrictor on his arm, I will briefly review the anatomy of the part. The tendons of the flexor sublimus and profundus cross the palm together. At the region of the metacarpo-phalangeal joint they pass under a firm ligament, the *ligamentum annulare-digiti*, and incased in a common sheath proceed down the finger. The tendon of the sublimus splits almost immediately, allowing the tendon of the deep flexor to pass through. The two parts of the sublimus tendon partially reunite and insert on either side of the second phalanx. The tendon of the deep flexor proceeds onwards to insert at the base of the terminal phalanx. Each tendon has two little additional insertions called the *ligamentum breve* and *longum*.

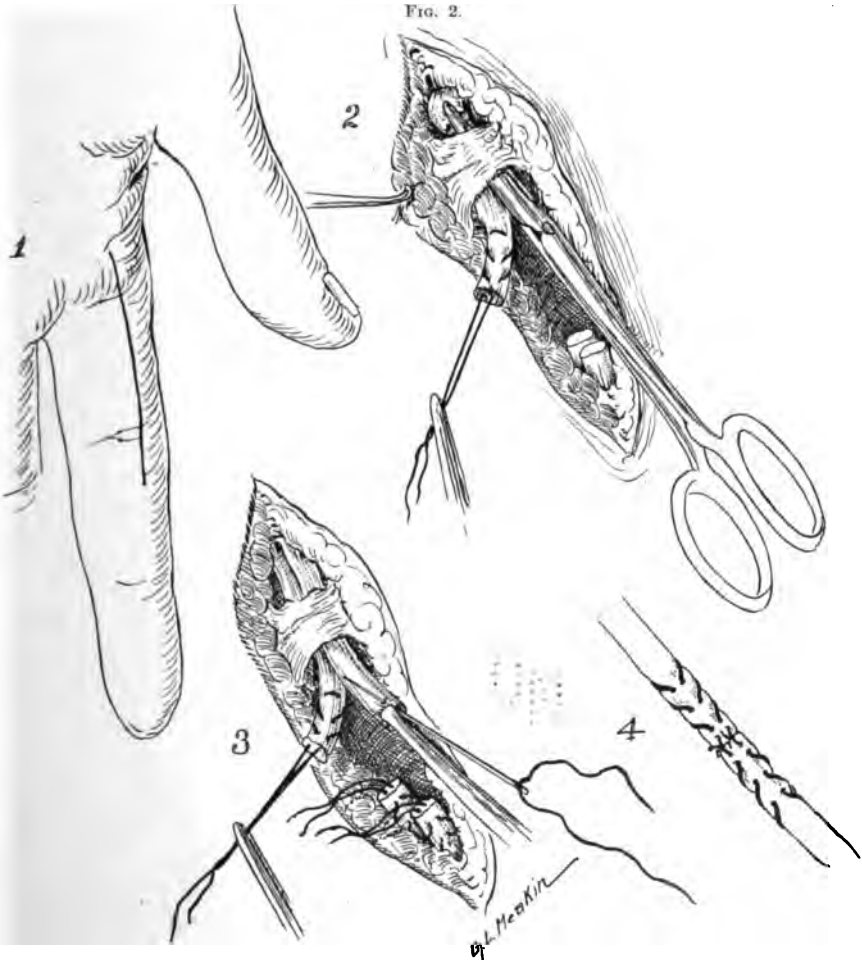
The dorsal interosseous muscle for the index finger arises from two heads, one from the ulnar side of the first metacarpal bone and the other from the radial side of the second metacarpal bone. These two small muscle bellies extend distally, join just below the level of the metacarpo-phalangeal joint and insert by means of a slender tendon into the dorsal expansion of the extensor tendon, the capsule of the metacarpo-phalangeal joint and the radial side of the first phalanx of the index finger. The palmar interosseous muscle arises from the ulnar side of the second metacarpal bone and inserts exactly as the dorsal interosseous except that it is on the ulnar side of the

FIG. 1.



A, Showing range of motion of injured finger. Notice, all the flexion is at the metacarpal phalangeal joint. B, Showing severed tendons, amount of retraction slightly exaggerated.

FIG. 2.



1, Skin incision, lateral in order not to be directly over tendons. 2, Passing severed tendons under annular ligament. 3, Placing silk sutures. 4, Method of placing sutures.

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phalanx. The lumbrical muscle is a small muscle slip which arises from the radial side of the deep flexor tendon in the palm of the hand and is inserted like the dorsal interosseous muscle on the radial side of the extensor tendon, on the dorsum of the phalanx (and also in the capsule of the metacarpo-phalangeal articulation). These small muscles arising in the hand and inserting on the dorsum of the phalanx and the extensor tendon naturally first approximate the phalanx to the palm and then, by pulling on the extensor tendon, extend the second and third joints.

The arteries of the index finger are four in number, two larger palmar arteries and two smaller dorsal vessels. The radial palmar artery comes from the deep palmar arch, the ulnar palmar artery from the superficial palmar arch. The radial dorsal artery is a direct branch of the radial artery, while the ulnar dorsal artery comes from the dorsal carpal branch of the radial artery. These four branches extend along the four corners (so to speak) of the finger. The dorsal digital arteries end at the base of the phalanx. The nerve supply is also by means of four main nerves, two dorsal from the radial nerve, two anterior from the median.

The patient is receiving a gas anaesthesia. A general anaesthesia is, I think, much to be preferred to a local one, provided, of course, no contra-indications to a general anaesthesia are present.

I am making my skin incision on the volar surface of the finger to the radial side of the midline (of the first phalanx) (Fig. 2). I am making a lateral incision because I do not want the scar directly over the tendon. This would offer more chance for the formation of adhesions. Also if I have to lengthen my incision proximally, as I probably will, the scar will not be pressing directly against the head of the metacarpal bone whenever the patient carries anything.

(This knife is laid aside and not used again.)

I am down to the tendon sheath, which I now open. Tracing the sheath upwards I come upon the severed end of a tendon. I will pull this well into view. We now see that this tendon splits. Note how one division leads to the right and the other to the left. I have, in other words, the distal end of the flexor sublimus tendon. I see that there is no tendon running through this bifurcation. The flexor profundus, therefore, must also be severed and retracted. Our diagnosis is confirmed. I grasp the superficial flexor carefully with a very fine mosquito forceps and will try to find the deep tendon. By

flexing the distal and middle phalanx I may be able to bring the end into view. This does not seem to suffice. I am carefully passing these light forceps distally into the sheath to try to locate the end in that way—but this also does not succeed. I must enlarge the slit in the tendon sheath. Here it is, the end has retracted and become attached to the side of the sheath. The adhesions are very easily loosened, and I now can pass this severed end of the deep tendon through the Y of the sublimus tendon. I grasp this also, gently, with a mosquito or seraphin clamp. I lay these two on the piece of gauze and start in search of the proximal ends. These may cause us more trouble. By extending the third, fourth and fifth fingers we make tension on the flexor tendons and thus pull the ends towards our incision. Pressure on the flexor surface of the forearm by the assistant also helps milk the tendons downwards, also flexion of the wrist. However, these manœuvres do not suffice. I will pass a small forceps under the annular ligament and see if I can grasp the elusive ends. I must enlarge my incision into the palm. I still keep to the radial side of the path of the tendons. I am very careful not to injure the annular ligament, because when I find the ends I am going to thread them under this ligament which will act as a pulley or more correctly as a bar. Sometimes when this ligament is cut the patient must wear a broad ring to act as the ligament to direct the pull of the tendons. I am now below the ligament, and you see the sheath is no longer well defined. Now, by extending the fingers, I see appearing at the very bottom of the wound a flat whitish band which looks like the proximal end of the superficial tendon; I grasp it gently and pull it into the wound and see that the whipcord-like deep flexor is pulled down with it. I feel that I am fortunate in having found both of the tendons so readily. At times they retract much further and at times become adherent.

The interne asks me whether it is necessary to locate both tendons. Usually in a case like this if we find one we find the other also, and if we can suture both our results will be much more like normal, and again we have two chances of having at least one flexor tendon uniting. After a severe infection we are often very contented to find one tendon. The deep flexor tendon gives us motion in an additional joint, the superficial tendon is the stronger of the two, due to its closer insertion.

I have threaded the found proximal ends under the annular ligament and now am ready for the actual repair (Fig. 3).

I am using small, fine, half-curved, non-cutting needles threaded with fine black silk. I am using silk because it is the strongest suture material for its size. It has the greatest tensile strength, as we used to say in physics. It will cause fewest adhesions. I feel sure that there is no infection present at the site of operation and that our technic has been good. If you have observed closely you have noticed that no fingers ever touched the wound. What little sponging has been done has been done with gauze on the end of a hæmostat, and for retracting and dissecting instruments, and not fingers were used. Split kangaroo tendon, or chromic catgut hold as well but are bulkier and although as satisfactory where we deal with larger, coarser tendons here in fingers are more apt to cause adhesions. Were there a real danger of infection I would, of course, not think of using silk.

While speaking I have placed the suture in the distal end of the deep tendon. I have made a double herring-bone stitch, starting on the outer side, leaving the end long, running upwards in three spirals, being careful to perforate between different fibres each time, perforating the tendon and then spiraling down on the inner side (Fig. 4). I have started and stopped about an eighth of an inch from the cut end so that I can trim this off. I am repeating the same procedure on the proximal end of the deep tendon and will again repeat on the proximal and distal ends of the superficial flexor.

The sutures are placed, and we are ready to tie. In order to relieve the tension as much as possible, we are flexing the wrist, extending the third, fourth and fifth fingers, and flexing the finger on which we are working. I am going to tie the deep tendon first. I am taking both outer ends and the first assistant both inner ends of the suture and simultaneously we slowly draw in on our first knot and approximate the two severed ends and place our second knot. I am now going to put in one small mattress stitch to reinforce these two lateral stitches. The ends of the sutures are cut, and the deep flexor tendon is repaired. We carefully tie the sutures of the flexor sublimus in the same manner. I am again putting in a reinforcement mattress stitch.

I am ready to close. The tendons have been united, the silk sutures are strong enough to hold until healing has taken place. We

have disturbed the anatomy as little as possible. The boy ought to have a good result. Before taking off the constrictor I will place one small catgut stitch in the tendon sheath to close it.

There are a few bleeding points which we will catch and ligate (with 00 catgut). The skin is closed with a continuous silk suture. The interne paints the wound with iodine and applies a sterile dressing.

I am going to put the hand on a splint. I am going to extend all the fingers except the index finger which is, as you see, in a position of semi-flexion from the tension of the united, partially retracted tendons. I allow this finger to remain in that position for the time being so as not to put too much strain on the repaired tendon. I will not disturb the dressing for three days. At the end of this time I will take out the skin suture and make slight passive motions. I will put aside the splint, protecting the finger by strapping it over a roller bandage. I will change this daily, using a larger and larger roll, thus overcoming the flexion we have now. I will start active motions about the fourteenth day, limited at first, gradually enlarging the range and decreasing the interval between exercises. The success of the operation depends in great part on the careful supervision of the after-care.

To sum up briefly the important points I wish to emphasize:

First: Do not be misled in your diagnosis in an injured finger by the action of the small muscles of the hand; they flex the first phalanx and extend the second and third.

Second: When immediate repair is impossible wait for all signs of infection to subside, and then wait a little bit longer.

Third: Make a lateral skin incision, not over the tendon.

Fourth: Injure the tendon sheath as little as possible, and be especially careful of the technic; be aseptic and be gentle.

Fifth: If you have difficulty in finding the proximal end of the severed flexor tendon extend all the other fingers.

Sixth: Spare the annular ligament if possible.

Seventh: In a clean case where delicate structures are to be repaired silk is the best suture material.

Eighth: The double herring-bone stitch is the least likely to pull out.

Ninth: Remember that the after-treatment is just as important as the operation. Too vigorous motions may tear out the suture; too long an immobilization will lead to the formation of adhesions.

## CLINICAL CONFERENCE ON OBSTETRICS

By BARTON COOKE HIRST, M.D.

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THE lecturer addressing the student conferring with him in the amphitheatre before the class says:

"The first thing to become familiar with is the routine questionnaire; please ask the usual questions of this woman, remembering that she has some condition peculiar to her sex."

The patient is a married woman of thirty, who had one child six years ago. Still alive and healthy. She says that the labor was difficult, but not instrumental. She had hemorrhoids, but there was no complication in the way of fever or infection which might have resulted in some pelvic adhesions.

In reply to the next question she denies ever having had a miscarriage. It is six years since the baby was born, so we need not inquire as to the duration of lactation.

The next question concerns menstruation. Beginning at the age of seventeen it was irregular and infrequent. Late and infrequent menstruation is significant of ill development of the sexual organs often associated with neurasthenia. Directly following the question about menstruation should come: What was the date of the last period? It was January 1st. It is now the end of March. This amenorrhoea of three months suggests pregnancy.

The next question, one that we are apt to forget if we do not keep it in mind, is: Has there been a previous operation? There has, but it was only a plastic operation. After some inquiry into the family history and previous diseases we ask: What is the chief complaint? We will anticipate a little and tell you that she is pregnant, and became so some time subsequent to the date of her last period. We compute the duration of pregnancy from the last period, as the only fixed date we have; but it is often forgotten that during the period, the patient was not pregnant. Impregnation occurs subsequently, usually just before the next period. Scientific investigation has demonstrated that pregnancy usually begins by the fertilization of the ovum of the first missed menstrual period. So if the woman's



last period began January 1st, she did not become pregnant January 1st or directly after, but most likely about January 28th. Therefore, it is incorrect to say, as we should have said in the past and are apt to say carelessly now, that such a woman is three months pregnant. It is a pregnancy of only two months.

To return to her chief complaint: it is excessive vomiting. When did you begin to vomit, and when did it become excessive?

*Patient:* Four weeks ago.

*Doctor Hirst:* We are carelessly apt to state that the nausea and vomiting of pregnancy begins at about six weeks. That is not accurate. As a matter of fact, these symptoms usually appear about two weeks after impregnation, but six weeks after the last period. We always have to subtract the length of the period from a possible pregnancy; and if we admit that a woman becomes pregnant, as a usual thing, just before the first absent period, we must agree that the nausea and vomiting begin two weeks after impregnation. They began, in this case, four weeks ago, which would be a little later. Four weeks ago would be the end of February. She presumably became pregnant about the 29th of January. Therefore, this nausea and vomiting appeared four weeks after the presumable impregnation, and eight weeks after her last period.

Here is an interesting fact, however, in the woman's history: She makes the statement that her menstruation is usually delayed and that the interval between her periods is about seven weeks. That works out according to the average, because it is likely that this woman became pregnant about seven weeks after the first of January. If she followed the general rule of becoming pregnant just before the first missed period, she probably conceived about the 18th of February. Therefore, the nausea and vomiting, which began about the first of March, appeared at the usual time.

This patient has applied to us in early pregnancy for what she describes as exaggerated vomiting. Let us get at the real extent of it. She says that everything comes up. That is the usual reply in such cases, but we always have to discount the statement of the patient. There are two things exaggerated by a patient, one is the amount of a hemorrhage and the other is the degree of nausea and vomiting. She says that everything comes up, but we cannot accept her statement unreservedly. We have observed the amount she has retained. As a

rule, in such cases considerable quantities of nutriment and liquid are retained, enough to justify a diagnosis of exaggerated nausea and vomiting, and not pernicious nausea and vomiting. It is necessary to draw a distinction between these two degrees of the same thing, because the conclusion drawn influences our treatment and prognosis. The former is a minor complication from the medical point of view, although distressing to the patient. Exaggerated nausea and vomiting is never fatal. It is extremely disagreeable, but never threatens life. On the contrary, pernicious vomiting is a dangerous disease, with a mortality of from 33 to 36 per cent.

This woman has been here twenty-four hours, and has not actually vomited in that time. Therefore, you see that her statement that she rejects everything is not accurate; for she has had at least two meals since she entered, if not three, and has not rejected them. She has constantly by her, however, a cup into which she is expectorating. In every patient who comes to us for excessive vomiting we must make sure whether what she is eliminating is vomitus or saliva. She is rejecting something now for our benefit. It is saliva. That cup given to the patient as she entered the clinic is already half full. So, doctor, from what is this patient suffering?

*Doctor:* Ptyalism.

*Doctor Hirst:* Here is a case sent to us by a physician and described by the patient as one of excessive vomiting; but we find that it is the other manifestation of the toxæmia of pregnancy, ptyalism, and not vomiting at all. She will fill this cup several times in the course of the day. I have seen this form of toxæmia much more exaggerated. I had a patient who, every night, had to have not a cup, but a slop-jar, by her bedside; and into this she would expectorate astounding quantities of saliva. She also went to bed with a box of marshmallows. She thought that eating them diminished the quantity of saliva, but I believe the constant chewing actually increased the expectoration.

Here we are dealing with marked ptyalism, as one of the early manifestations of the toxæmia of gestation. It is perhaps an effort to get rid of the toxins by means of the salivary glands.

Now what should we do? That is, after all, the aim of all our investigations. What would you recommend for a case like this in a private patient?

*Answer:* Increase elimination through other channels.

*Doctor Hirst:* But what would you do? You cannot cultivate too early the habit of giving precise and definite directions. That is one of the things that success in practice depends upon. You should give definite directions. What should they be? You have a perfect right to your opinion as to what should be done, but you must acquire the habit of giving precise directions. What would you tell the family or the nurse with the idea of increasing elimination by other channels than the salivary glands?

*Answer:* Take lots to drink.

*Doctor Hirst:* If called in consultation, I should venture to disagree with such a recommendation. I think it would probably increase her ptyalism. What else can you think of to control this symptom? You will be asked to prescribe for it quite frequently. It is very inconvenient, to say the least; and there is a certain drain on her strength from the constant elimination of large quantities of saliva. The woman is never made as weak as she would be if she were vomiting incessantly, but she is reduced in vitality and strength. She wants relief. What can you give?

*Answer:* Atropia.

*Doctor Hirst:* That is a remedy to be considered. Naturally, we have to be cautious about administering large quantities of a powerful drug to a pregnant woman on account of the effect on the embryo; but the embryo does not appear to be susceptible to atropia. We could give full doses of that remedy and it is a sensible suggestion. Is there any other drug that would not only decrease secretion but would control one of the elements of toxæmia? One element of this so-called toxæmia of pregnancy is neurosis, another is toxæmia, and a third is reflex irritation. Is there another remedy that would occur to you that would diminish secretion and, at the same time, control one of these elements of toxæmia, neurosis? In ptyalism there is always a strong neurotic element, so we are inclined to attribute to the neurotic element in ptyalism more importance than we do in vomiting. What drug would suggest itself to you as applicable in a case of this kind?

*Answer:* Morphia.

*Doctor Hirst:* Morphia may be considered. It would diminish secretion and control nervousness, but there is a natural disinclination to administer opium for any length of time. Physicians fear the

formation of a habit. We should avoid the routine use of opium in these cases; although in individual cases, if other things fail, we may think of it as an appropriate remedy.

Chloral has been of use in such cases. A moderate dose of chloral, ten grains, twice or three times a day, well diluted, with a moderate amount of atropia, would be the most suitable remedy to administer.

Moreover, we have the chance of a spontaneous improvement as we have in cases of excessive nausea and vomiting. Ptyalism rarely persists during the second half of the pregnancy, so if the treatment is not successful, we can hold out the hope that the condition will ameliorate itself in the course of time. It is never fatal.

A gentleman very properly asks concerning reflex irritation. We do not find by clinical experience that reflex irritation causes this symptom. We, therefore, consider only two of the three elements of the toxæmia of pregnancy; the actual toxæmia and the neurosis, which is always well marked in these cases.

#### SECOND PATIENT

What is the age of this patient? Twenty-eight.

Has she had any children? No.

How long has she been married? Ten years.

Have there been any miscarriages? No, she has never been pregnant. Her menstruation began at the age of sixteen years. She says it is regular, occurs every twenty-eight days, lasts four days. The quantity is never great and is sometimes scanty. The last period was two weeks ago. There is leucorrhœa. She has pain several days before the period, ceasing when the flow appears. What does that indicate, as a rule?

*Answer:* Obstruction.

*Doctor Hirst:* No. It suggests inflammation. It is typical of salpingitis. With that idea in mind, when a patient makes such a statement, we ask: Is the pain worse on one side or the other, or is it in the middle line? If a woman has salpingitis on both sides, she feels it worse on the left.

This patient has regular, but scanty menstruation, with pain two or three days before each period, worse on the left side and relieved by the flow. That is usually indicative of salpingitis.

Has there been a previous operation? No.

We come now to the patient's chief complaint. She complains of a tumor, or, as she describes it, of a lump in the abdomen. I have to report the physical findings as we cannot make these examinations in public. We find an irregular shaped tumor filling the lower abdomen and reaching nearly to the umbilicus, the greater bulk of which is on the left side. It is firm, and is attached to the uterus; and it moves with every movement of the uterus. What would be your diagnosis?

*Answer:* A fibroid tumor.

*Doctor Hirst:* And what kind of a fibroid, in view of the fact that the woman has scanty menstruation, would you think it to be?

*Answer:* Subperitoneal.

*Doctor Hirst:* Yes. If it were submucous she would bleed more. What would you recommend for treatment? I am addressing an audience of prospective general physicians. I do not purpose to ask about the technic of surgery. But if a patient applies to you and you find what we find here, what would be the alternative recommendations for treatment? What must the general physician consider?

Well, he might recommend an operation and refer the patient to a surgeon, if he could not do it himself; but what must he consider besides the operative treatment?

*Answer:* He can use radium.

*Doctor Hirst:* That is the other plan; and what other agent is thought to be even better than radium by the real experts in this sort of treatment?

*Answer:* The X-ray.

*Doctor Hirst:* Operation implies removal by one way or another. The alternative to operation is the X-ray or radium or both combined. I might say a word on the choice from the point of view of the general physician, or my own, for that matter. If I think that a patient is not a good surgical risk, is not suitable for myomectomy, or is of an age that makes the premature menopause resulting from X-ray or radium a matter of indifference, I send her to a röntgenologist, who is competent to decide which is the better treatment; and I find that those familiar with both methods usually prefer the X-ray. A gynecologist who has bought radium as an investment and must get an adequate return from it is inclined, naturally, and sometimes unconsciously, to the excessive use of radium, and is not equipped to make an intelligent choice between radium and the X-ray. It seems to me

that the latter is the better treatment for a case like this. Radium put into the uterus would be filtered by the tumor, which would prevent its having full effect upon the ovaries. The X-ray would have only the abdominal walls to filter through. A long and tortuous uterine canal would prevent the action of the radium on the whole extent of the tumor and the whole area of the endometrium. I find that specialists accustomed to both, prefer the X-ray to radium and I think they show common sense.

In the case of a private patient of intelligence, who can keep herself under the observation of a physician for a length of time, and in whom there is a moderate sized fibroid tumor that is not productive of serious symptoms, what would you advise?

*Answer:* If she were near the menopause, I would leave her alone.

*Doctor Hirst:* Yes, and keep her under observation. I had a case in my office yesterday. This patient was brought to me by her physician three years ago. She had a moderate sized fibroid the top of which reached within three fingers' breadth of the umbilicus. She had nothing to complain of, except a few minor pressure symptoms. She had a bad neurotic family history, so that any operative procedure of even slight character might have been followed by post-operative mania, and certainly neurasthenia, or an exaggeration of the neurasthenia that she already has. I advised that her physician keep her under observation, and that she come to the specialist's office at least once every three months. I saw this patient for the last time yesterday, three years after my first examination, and found that in the course of these three years, the tumor had increased in height by the breadth of two fingers and correspondingly increased in bulk in other dimensions. The woman's condition was as it had been for three years before. There was no menorrhagia or metrorrhagia, but the pressure symptoms were more marked. So her doctor and I decided to refer her to an expert in röntgenology and have him apply the X-ray treatment. Radium would necessitate some little operative intervention, in order to insert the tube into the uterus; and that we thought it best to avoid. We decided on the X-ray treatment as in all probability, being best, but left the choice between it and radium or the combination of the two to the röntgenologist to whom we referred her. I think that you will find in your future practice that you will be taking that course more frequently than any other in the man-

agement of fibroid tumors. What would justify you, as a general physician, in recommending operative treatment?

*Answer:* If the tumor is of excessive size or causes serious pressure symptoms.

*Doctor Hirst:* Yes, whether the size is great or not, the tumor may be blocked in the pelvis and be so low as to make serious pressure on the rectum and bladder, irrespective of its size. But it must be remembered that X-ray and radium may shrink the tumor and so relieve the symptoms.

*Another Answer:* If she wants to have children.

*Doctor Hirst:* Yes. Sterility is a consideration that should influence us. The removal of fibroids by myomectomy in women of child-bearing age is followed in 18 per cent. of the cases by conception; showing the fibroid to be the cause of the sterility. X-ray and radium in such cases would prevent conception by establishing the menopause. Now suppose that you have a moderate sized tumor with excessive bleeding, and that there are reasons why you do not consider operative treatment advisable, and there may be difficulty in securing X-ray or radium treatment; so you decide to try medicinal treatment. What can you prescribe? Whatever would be good in such a case would be useful in other cases of menorrhagia or metrorrhagia, so it is well to have in mind a routine prescription. What would you prescribe?

*Answer:* Some astringent might be useful.

*Doctor Hirst:* Never put it that way. Imagine yourself in actual charge of this case. The woman bleeds too much, and you have made up your mind to try the medicinal control of the bleeding. Therefore, you write a prescription and give it to your patient. One of the most useful prescriptions that a doctor can have is one for the control of uterine bleeding. There are several prescriptions that a physician must be ready to scribble off automatically; and one of these is this. If you are asked to prescribe for a woman who is bleeding from the uterus, either periodically or continuously, and you will have this kind of case over and over again—what will you give?

*Answer:* Strychnine sulphate.

*Doctor Hirst:* Of course, as a doctor, you can write anything you like, but it should be effective. Strychnia would not stop hemorrhage. What might you give?

*Answer:* Calcium lactate, to increase the coagulability of the blood.

*Doctor Hirst:* I find the following more efficient: A grain of ergotin, a grain of stypticin, and half a grain of hydrastinin, in pill form, four or five times daily.

There is another thing we must consider in this connection, something that you will have to take into account almost every day in your future busy office practice or visits to your patients' homes: Suppose a patient complains of menorrhagia or metrorrhagia, you exclude fibrosis of the uterine muscle, a fibroid and cancer and conclude that it is from the endometrium, from some cause you cannot at once determine. What must a doctor be on the lookout for?

A woman comes to my office and says that she bleeds excessively either continuously or periodically. I exclude the commoner causes of such bleeding, find nothing very definite in the examination; and decide that the bleeding comes from the endometrium; from some cause not immediately apparent. What should one do?

*Answer:* Make a complete examination.

*Doctor Hirst:* Of what?

*Answer:* The blood pressure.

*Doctor Hirst:* Yes; always take the blood-pressure and listen to the heart. A young unmarried girl was sent to me for metrorrhagia. I found the blood-pressure 210 systolic; her metrorrhagia was due to this cause alone. I have sometimes made the mistake of treating metrorrhagia in vain till a heart murmur was discovered. What else may cause it?

*Answer:* Syphilis.

*Doctor Hirst:* Yes, a Wassermann reaction is part of the routine examinations for the cause of metrorrhagia. What other routine examinations must be made?

*Answer:* The examination of the blood for malaria.

*Doctor Hirst:* Yes. What else do we investigate?

*Answer:* The clotting time of the blood.

*Doctor Hirst:* Yes. We investigate the blood for syphilis and malaria and the clotting time of the blood.

Take a woman's blood-pressure; listen to her heart; have a Wassermann and take the clotting time of the blood in every obscure case of uterine bleeding.



If all these examinations are negative, if medicinal treatment fails, and the woman is too young to risk a premature menopause by radium or the X-ray, are there any local applications that will stop the hemorrhages?

*Answer:* Yes, iodine might.

*Doctor Hirst:* No, you would be disappointed if you tried it. Three agents are to be thought of: the positive pole of a galvanic current, with an intra-uterine electrode 60 milliamperes or more; 15 per cent. solution of formalin packed in the uterus on a strip of gauze; or a 10 per cent. solution of chloride of zinc.

## A SURGICAL CLINIC

AT THE MEDICO-CHIRURGICAL HOSPITAL

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### ULCER OF DUODENUM; POSTERIOR GASTROJEJUNOSTOMY

A. O., male, white, aged thirty-one, tinsmith, complained, about one month previous to admission, of radiating pain throughout abdomen, severe enough to require a dose of morphia. He was not confined to bed during the attack. Six days previous to admission he had another attack, which also required a dose of morphia. His appetite has been good. He has not been constipated. He has not belched. He has not lost weight. On taking a deep breath there is "wincing" pain in the right hypochondriac region. Physical examination of heart and lungs negative. There is no jaundice. Examination of abdomen reveals no muscular rigidity. Palpation over "gall-bladder area" elicits tenderness. A fluoroscopic examination was made, but the report was negative.

*Operation* (September 25, 1920).—Ether anæsthesia. Vertical incision through middle of right rectus, supraumbilical portion. On exploration an adhesion band was found, with lower end attached to middle of anterior surface of first portion of duodenum, length about 7.5 cm., and with upper end attached to liver to right of gall-bladder. This adhesion compressed summit of duodenum: it was dissevered. At the duodenal attachment of the adhesion band there was found a white scar suggesting ulcer, and palpation revealed thickening of the wall of the duodenum at this site. The gall-bladder was distended with bile, but was soft, supple and compressible. The stomach was somewhat dilated. Posterior gastrojejunostomy was performed, aided by a Barrett clamp, using linen for the external coats, and No. 0 chromic gut for the internal. The ostium was made at the most dependent point of the stomach: it was  $1\frac{1}{2}$  inches in length, and oblique in direction (*i.e.*, from above and to left, downward to right). The jejunum was adjusted by the "no loop" method. Angles of anasto-

mosis were reinforced, and gap in mesocolon was closed by suturing latter to stomach. All sponges and instruments accounted for. Abdominal wound closed without drainage by the tier method.

*Note* (October 5, 1920—ten days after operation).—Wound healed *per primam* throughout. Patient has been relieved of all symptoms and can eat anything without discomfort. He will be sent home to-day.

*Comment.*—The interesting features of this case are, first, the atypical history of duodenal ulcer, although the patient did not speak English well, so that the history was not entirely satisfactory. The history does suggest, however, in the first attack one month previous to admission, wherein the patient complained of “wincing” pain in the right hypochondrium when taking a deep breath, that this was the beginning of the formation of the adhesion that was found at operation. The second attack, six days previous to admission, was probably due to a recurrence of localized peritonitis in the vicinity of the adhesion band. When stomach and bowel were opened during the performance of the anastomosis both cavities were found to contain bile, whose presence was accounted for by compression of the gall-bladder during the course of the exploratory examination. Drainage of the stomach must have been good from the beginning, because the patient did not vomit after operation, despite the presence of the bile in that organ, nor was he even nauseated. Note that the pylorus was not intentionally occluded, as advocated by Strauss and others. The presence of dilatation of the stomach suggested a certain degree of pyloric obstruction already present, although the patient at no time vomited previous to operation, and this dilatation perhaps accounted for his smooth, rapid, and complete recovery in the absence of intentional pyloric blockage.

STONES IN COMMON BILE DUCT; CHOLECYSTITIS; CHOLEDOCHOTOMY;  
CHOLEDOCHOSTOMY; CHOLECYSTECTOMY

O. R., female, white, aged forty-seven, has been complaining for the past twenty-one years of pain in the right upper abdomen, the attacks occurring once or twice a year. Every three or four days she is attacked by pain in this region. This pain does not radiate to right shoulder. She suffers from dyspepsia—acid eructations, gas on stomach (belches), food lies like weight on stomach. She is consti-

pated, her bowels moving every two or three days. She has lost weight in the last few months. During the attacks of pain she claims that her skin becomes of a yellowish hue. Physical examination reveals an icteroid tinge of the conjunctivæ and skin. The heart beats strongly and regularly, and the lungs are clear. Examination of abdomen reveals "wincing" tenderness to finger percussion test (Murphy) over gall-bladder area. There is no muscular rigidity. Vaginal and rectal examinations are negative.

*Operation* (September 7, 1920).—Using novocain 1 per cent. the Bevan S-incision was made through abdominal wall, right rectus. Gall-bladder found distended; common duct found to contain stones. Ether was now begun. No adhesions were encountered. Stomach and bowels walled off by gauze packs. Packs placed in renal pouch. Common duct brought into field and incised between two silk stay sutures: much bile escaped and was removed by mopping. Six large gall-stones size of end of thumb were milked out through opening. Common duct was found dilated to size of index finger. Using a large flexible probe the common hepatic and common bile ducts were explored and found free from additional stones. Cystic duct isolated and divided between two clamps. Cystic artery identified, separately ligated with catgut, and divided. Gall-bladder removed: it was distended, but contained no stones. Gall-bladder fossa closed by suturing cut peritoneal edges on each side. A rubber drainage tube  $\frac{1}{4}$  inch in diameter was split and sutured into common duct: incision in common duct closed up to tube. A No. 14 F rubber catheter and cigarette drain were placed in right renal pouch of Morison. Packs removed. Instruments accounted for. Duct drainage tube brought out through suspensory ligament of liver and, with other drains, through middle of wound. Sandbag removed. Instruments and sponges accounted for. Incision closed by tier sutures.

*Note.*—Five days after operation patient was transferred to another ward, and while in transit the tube slipped out of the bile duct. Ten days after operation all drainage was removed. Thirteen days after operation bile no longer escaped from the fistula. Sixteen days after operation the sinus left remaining was almost healed, and by this time the icteroid had cleared away. Patient discharged, cured, on the sixteenth day after operation.

*Comment.*—Despite six large stones in the common duct the

patient was only icteroid: this suggested a bile-way between the stones and the wall of the bile duct. Furthermore, there was no pancreatitis. Courvoisier's law was violated, or it did not operate because the gall-bladder was distended. The history was fairly typical of disease of the biliary system. The patient was fair and forty, but not fat. The convalescence was smooth, rapid, and complete, the temperature at no time rising above 99° F. The biliary fistula closed rapidly (thirteenth day). This rapid closure seemed to have been greatly aided by the method of bringing the duct drainage tube out through a hole in the suspensory ligament of the liver, and suturing this ligament down upon the bile duct around the tube, thus effectually sealing a potential leakage point and obliterating the fistula by rapid peritonealization. This procedure is believed to be original, although the writer got the idea of it from Dr. Charles Mayo's method of interposing the great omentum between the bowel and abdominal wall in performing jejunostomy: the omentum is perforated by the ostomy tube, and the resulting fistula closes more rapidly when the tube is removed. The split rubber tube as a substitute for the T-tube in choledochostomy does not seem to have been emphasized elsewhere.

**INCARCERATED FEMORAL HERNIA COMPLICATED BY DIABETES MELLITUS: HERNIOTOMY; REDUCTION; HERNIORRHAPHY, USING LOCAL ANÆSTHESIA**

R. J. T., female, white, aged seventy, was admitted on September 9, 1920, with an irreducible swelling the size of two fists in the right groin. Three years previous to admission, after an accident, the patient developed a right femoral hernia. This hernia was comparatively small and freely reducible until one week previous to admission when, during the strain of lifting, it suddenly became larger and could not be reduced. The patient since then has been nauseated, but did not vomit. Her bowels have continued to move. Physical examination of the right groin reveals a tumor the size of two fists, irreducible and with no impulse on coughing, and apparently emerging from saphenous opening. The vascular system is the seat of marked arteriosclerosis. Examination of urine reveals the presence of sugar. The patient is a poor risk for ether, and therefore local anæsthesia will be used.

*Operation* (September 10, 1920).—Infiltration with novocain, 1 per cent. Incision made as for inguinal hernia, to which was added a vertical incision descending over mass and involving superficial tissues. Hernial sack exposed and found tense, cystic and globular, and with its pedicle passing through the saphenous opening. Hernial sack incised: dark serum escaped. Contents of sack found to be matted omentum surrounding the cæcum. Femoral ring and neck of sack exposed through inguinal canal. Constriction of neck of sack divided. Matted omentum ligated by transfixion and cut away: stump replaced in abdominal cavity. In order to replace cedematous cæcum it was found necessary to divide Poupart's ligament. Cæcum could now be reduced. Sack ligated high up and cut away. To prevent recurrence the round ligament with cremaster muscle and fascia still attached and intact was sutured to pectineus fascia, obliterating inner compartment of femoral canal. Poupart's ligament sutured at point of division. Superficial tissues and skin closed. Drainage of subcutaneous spaces, consisting of several pieces of gauze and rubber dam, inserted. Dry gauze dressing.

*Post-operative Notes.*—Six days after operation all drainage was removed. Eight days after operation all sutures were removed. Fourteen days after operation the patient was allowed to get up out of bed. Sixteen days after operation the patient was discharged, cured. Under the Allen treatment sugar disappeared from urine within a few days.

*Comment.*—Why was there no intestinal obstruction in this case of incarceration of one week's standing? Because the ileum at its junction with the cæcum was not drawn into the constricting ring, but rested upon the upper surface of Gimbernat's ligament as upon a shelf, constituting, in fact, a Richter hernia on a large scale. The presence of the cushion-like omentum in the sack, the thick wall of the cæcum and the absence of a mesocæcum probably prevented the cæcum from becoming gangrenous. Nor was the appendix, which also lay within the sack, gangrenous. The method of suturing the round ligament with the attached and intact cremasteric muscle and fascia to the pectineus fascia to prevent recurrence is believed to be original. The finding of sugar in this aged woman the subject of marked arteriosclerosis shows the advantage of making a complete examination in emergency cases before administering ether. It will be admitted that

the idea of employing the cremasteric muscle and fascia in femoral hernia was obtained from Halsted's use of it in the repair of inguinal hernia, but in a somewhat different manner.

STRANGULATED INGUINAL HERNIA: HERNIOTOMY; REDUCTION;  
HERNIORRHAPHY, USING LOCAL ANÆSTHESIA

B. D., male, white, aged fifty-one, was seen in consultation and immediately sent to hospital as an emergency case on August 19, 1920, at 7.30 P.M. The patient has had a right inguinal hernia since he was five years of age. He has worn a truss for about nine years. On day of admission, at noon, he became sick soon after eating and vomited considerably. He had another attack of vomiting about 4.00 P.M. His bowels did not move in this short time. Physical examination revealed in the right inguinal region an irreducible mass the size of a foetus head at term. The patient recovered from an attack of typhoid fever one month previous to admission to hospital.

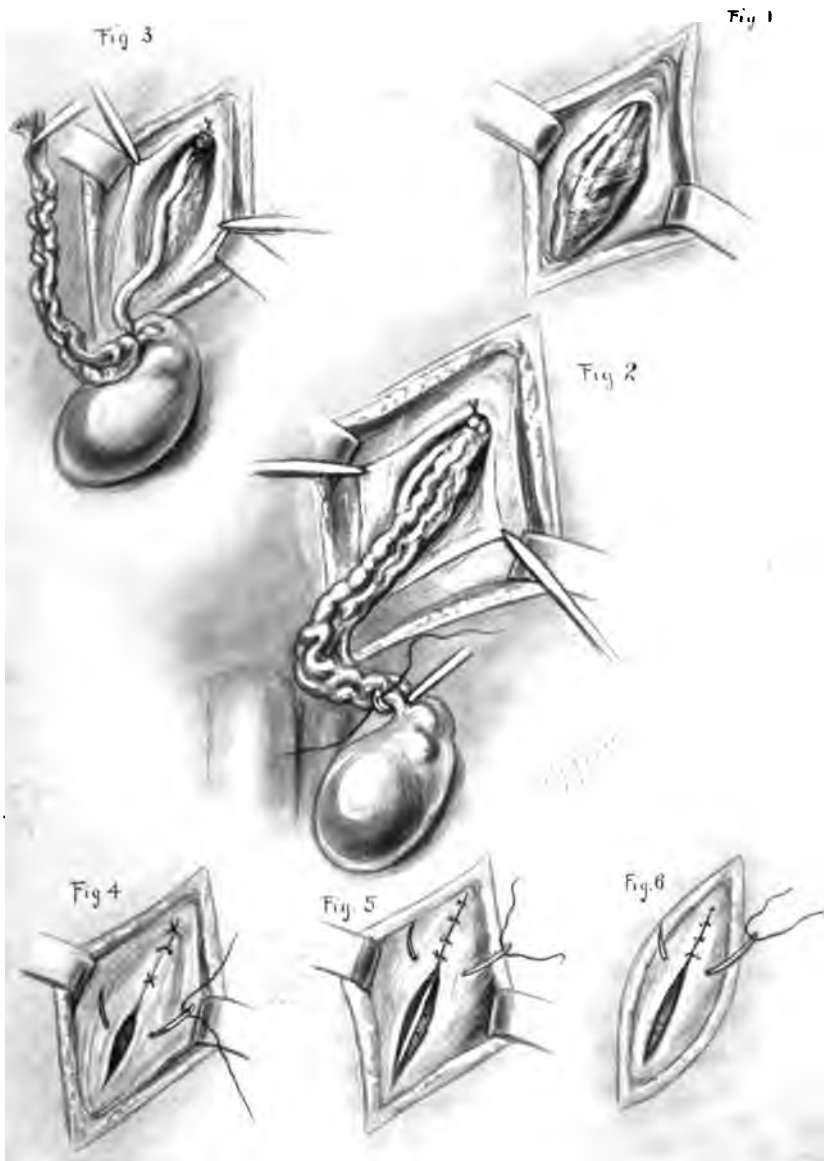
*Operation* (August 19, 1920).—Infiltration with novocain, 1 per cent. Incision over mass, above and parallel with Poupart's ligament. Superficial tissues divided; sack exposed. Sack opened: contents a dusky knuckle of small bowel, entirely outside of external ring. Large quantity of serous fluid, fresh fibrinous adhesion formation, œdema of peritoneal tunic of bowel. Contents reduced. Hernia repaired by modified Bassini method. Wound closed without drainage. Dry gauze dressing.

*Post-operative Notes*.—August 26th: Sutures removed, wound healed *per primam* throughout.

September 4th (sixteen days after operation).—Discharged, cured.

*Comment*.—This patient was operated upon eight hours after strangulation commenced, and with the usual favorable outcome when these cases are gotten early. The pathology was in consonance with the short duration of strangulation. The writer takes this opportunity of reiterating his oft-expressed opinion, that the way to secure a favorable outcome in a case of obstruction of the bowel, aside from getting the case early, is to operate under local anæsthesia. If you are not dealing with an external hernia be satisfied with enterostomy and drainage as your first procedure, ridding the patient of the lethal intestinal toxins and later doing the radical work. If you do not

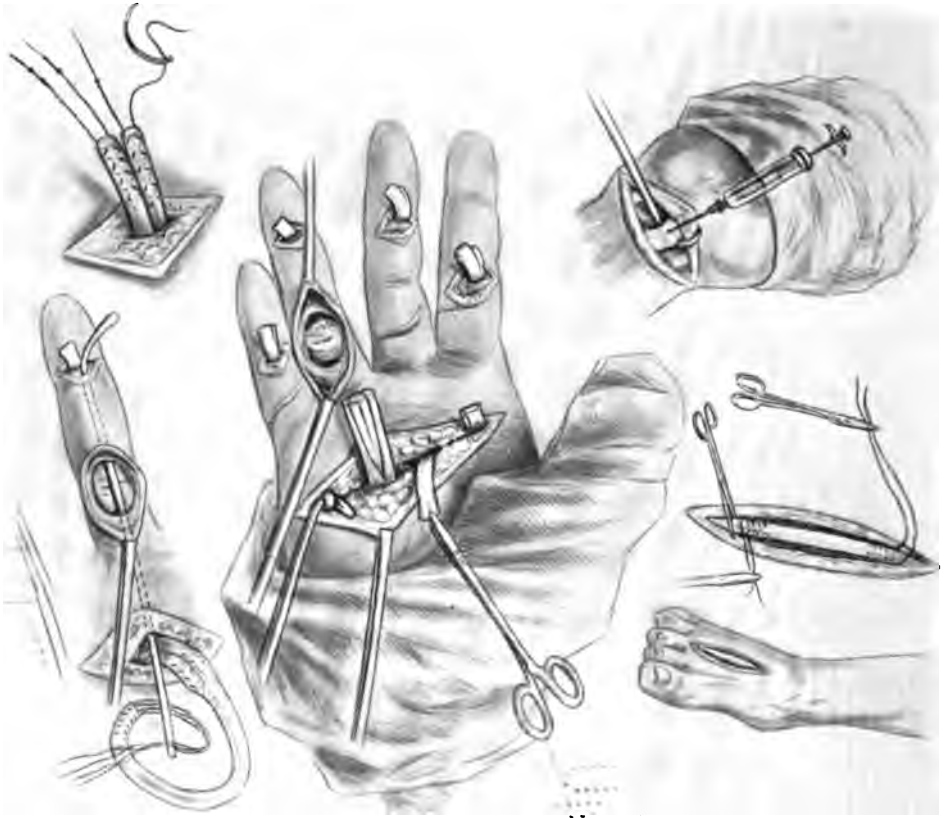
PLATE I.



The author's varicocele operation. 1. Exposure in inguinal canal. 2. Ligation above at internal ring and below at testicle. 3. Incision at internal ring, showing vas situated posteriorly. 4. Closure of cremaster muscle and fascia. 5. Closure of external oblique aponeurosis. 6. Closure of Scarpa's fascia. Steps 4, 5 and 6, obliterate dead spaces and check oozing.



PLATE II.



Shows steps in transplanting tendons of extensor digitorum longus from foot to flexor digitorum longus tendons, which had been severed by a cut across fingers. Note "feather" suturing and blocking of nerves. The distal ends of the transplants must be sutured to the periosteum of the inguinal phalanges, for the distal ends of the original tendons would undergo necrosis from insufficient nutrition.

know where to establish your enterostomy establish it in the jejunum and let reverse peristalsis, which is part and parcel of complete bowel obstruction, do the rest. If you have definitely established the large bowel as the seat of obstruction, perform cæcostomy under local anaesthesia and stop right there. If you have seen these obstruction cases go bad on the table under ether it is because you are adding ether toxæmia and ether shock to the intestinal toxæmia already present in these advanced cases, and perhaps because you are more concerned with doing a "complete job" by removing the obstruction than with the safety of the patient, which is almost ensured by enterostomy. In your next case try simple enterostomy under local anaesthesia and watch your patient get well.

#### OPERATION FOR VARICOCELE

The accompanying illustrations (Plate I) show the writer's operation for varicocele, which was presented before the Philadelphia Academy of Surgery on April 5, 1920, and a description of which will be found in *Annals of Surgery* for October, 1920, p. 508. You open the inguinal canal and free and remove the entire anterior vein mass from internal ring to top of testicle, carefully avoiding the vas and its deferential vessels. Thus, you remove the entire pathology. This operation is especially designed to avoid that column of induration so commonly encountered after certain other methods. When closing up obliterate all dead spaces. Perform the operation under local anaesthesia—ether is not justifiable. Avoid a compressing dressing over the external ring region: it will favor cedema of the scrotum and hydrocele formation. Have the patient wear a suspensory, beginning immediately the operation is terminated. He may leave hospital on the fifth day.

#### TENDON TRANSPLANTATION FOR DIVIDED FLEXOR TENDONS OF FINGERS

The accompanying series of sketches (Plate II) shows the various steps of an operation the author found necessary to employ for a patient who had grasped an adversary's knife edgewise. The tendons were not sutured primarily and consequently drew up within their sheaths beyond the possibility of restoration. An extensor longus digitorum tendon was taken from the dorsum of the foot for each of

the four fingers. The steps of the operation and method of suture are well shown in the illustration. You will also note that the operation was performed under local anæsthesia, exposure and blocking of the median nerve and exposure and blocking of the ulnar nerve being well shown. The tendons are transplanted with their sheaths. The suture material is No. 0 chromic gut. Silk should not be used for fear of infection with sinus formation.

ACUTE SUPPURATIVE APPENDICITIS: APPENDECTOMY, USING LOCAL  
ANÆSTHESIA

J. R., male, white, aged thirty-eight, bookkeeper, was seen in consultation on October 23, 1920, at 6 P.M. He had had three attacks of acute appendicitis, and this was the fourth. This attack began twenty-four hours previously with pain in R. I. F., not radiating and not followed by vomiting. Bowels moved after a laxative. Physical examination revealed exquisite superficial tenderness over R. I. F., greatest near A. S. S., with rigidity localized to same region. Temperature  $99\frac{1}{5}^{\circ}$  F.; pulse, 90. Rectal examination negative. Examination of heart and lungs negative.

*Operation* (October 23, 1920, 8:30 P.M.).—Morphin-hyoscine narcosis. Local infiltration anæsthesia, using procaine 1 per cent. McBurney incision. Peritoneum opened close to its fornix, turbid serum escaping. Appendix behind and to outer side of cæcum, greatly congested and distended to size of man's thumb—no perforation. Meso-appendix ligated. Appendix ligated at junction with cæcum, using No. 2 chromic gut, clamped and removed. Stump was not buried. No drainage. Wound closed.

*Comments.*—Here is the kind of a case we all like to get early. The appendix was tensely distended with pus and threatened to "explode" at any moment. With the organ in this position, namely, behind and to the outer side of the cæcum, the hazard was not so much that of diffusing peritonitis, as retroperitoneal cellulitis, which is much less amenable to treatment. It was gratifying to perform the operation under local anæsthesia because at the end of the operation one feels quite sure of the patient's uninterrupted recovery, provided the local infection is wholly eliminated, which in this case seemed assured.

## SURGICAL CLINIC

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We have in this case to deal with a patent urachus. The origin of this condition can best be understood if we refresh our memories as to the embryological development involved in this malformation.

The allantois first presents as an endodermal diverticulum which pushes itself into the mesoderm forming the belly-stalk (see Fig. 1). That portion of the allantois which is to be found in the umbilical cord persists until birth in a more or less rudimentary form, while that portion within the embryo undergoes many changes. The lower portions expand to form the urogenital sinus and the urinary bladder. The portion intervening between the apex of the bladder and the umbilicus usually degenerates into a solid cord of fibrous tissue termed the urachus.

Occasionally the urachus remains partially or completely patent. Where the urachus is completely patent, the urine passes from the bladder through this channel and is discharged at the umbilical opening. The partially patent forms are of three types. The first type presents a canal which extends from the navel downward for a variable depth toward the bladder. The second type presents a canal extending as a prolongation upward of the apex of the bladder toward the umbilicus. The third type develops as a dilatation of the lumen of the urachus with cyst formation.

The condition of patent urachus is found about four times as commonly in men as in women. It is usually congenital, but may be acquired where there is present a predisposition to patency as a result of incomplete atrophy of its structure. In such acquired cases the determining cause is increased back pressure of urine within the bladder due to strictures or other obstructions to the free outflow through the urethra.

The presence of small calculi within the lumen of the fistula has

been mentioned by several writers. These calculi have, as a rule, nuclei of foreign material coated over with urinary salts.

#### REPORT OF CASE

Mr. J. W., a switchman, thirty-seven years old, entered the hospital complaining of pain, swelling, and purulent discharge from the umbilicus. Three weeks previous to admission he noticed that his abdominal wall was sore and tender about the navel. With pressure he was able to express some pus from the umbilicus. The application of various poultices seemed to give little relief, so he decided to come to the hospital for treatment.

Ten years ago the patient states that he had a serous discharge from the umbilicus, but this ceased in a few days. Patient denies syphilis. He admits of having had four infections of gonorrhœa.

*Physical Examination.*—Examination was negative except for a swollen, red, tender, indurated area around the umbilicus, from which there was a purulent discharge. There was no evidence of peritoneal involvement. The urine showed no pathological elements on examination.

*Course.*—The patient had hot dressings applied every four hours for six days, during which time he had only a very slight fever. On the seventh day, under ether anæsthesia, the area about the umbilicus was incised. Hot fomentations were again applied for several days when the temperature returned to normal. Seventeen days after admission the infection had entirely cleared up, but it was deemed advisable to wait at least another week before doing the radical excision of the fistula.

*Operation.*—Under ether anæsthesia I make an elliptical incision to one side of the umbilicus carrying it down to the fascia of the rectus muscle. I now open the abdomen through the linea alba above the umbilicus. On opening the peritoneum and inserting retractors you can easily make out the prominent median peritoneal fold passing upward from the bladder. On either side you see these two smaller folds which cover the obliterated hypogastric arteries (Fig. 2 (1)). Using sharp dissection, I now encircle the umbilical area, using a technic similar to that used in dealing with a fecal fistula of the abdominal wall. Having freed the urachus at its distal end, I now dissect it free toward the apex of the bladder and apply crushing

FIG. 1.

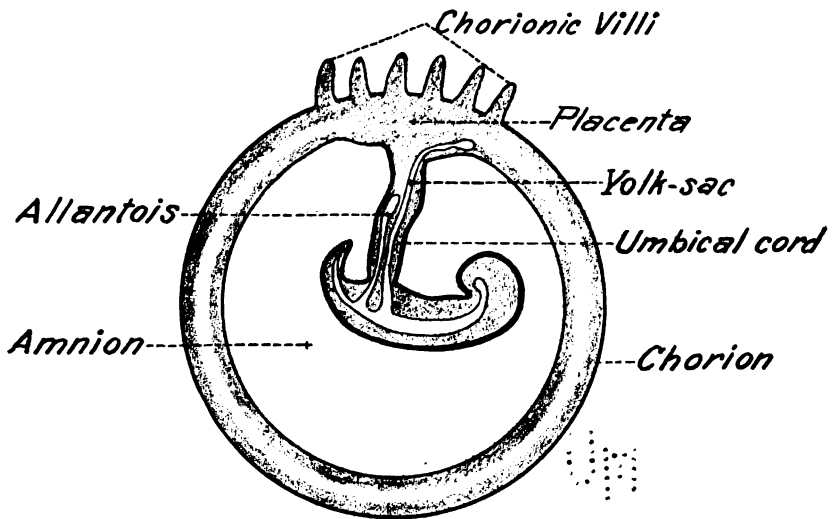
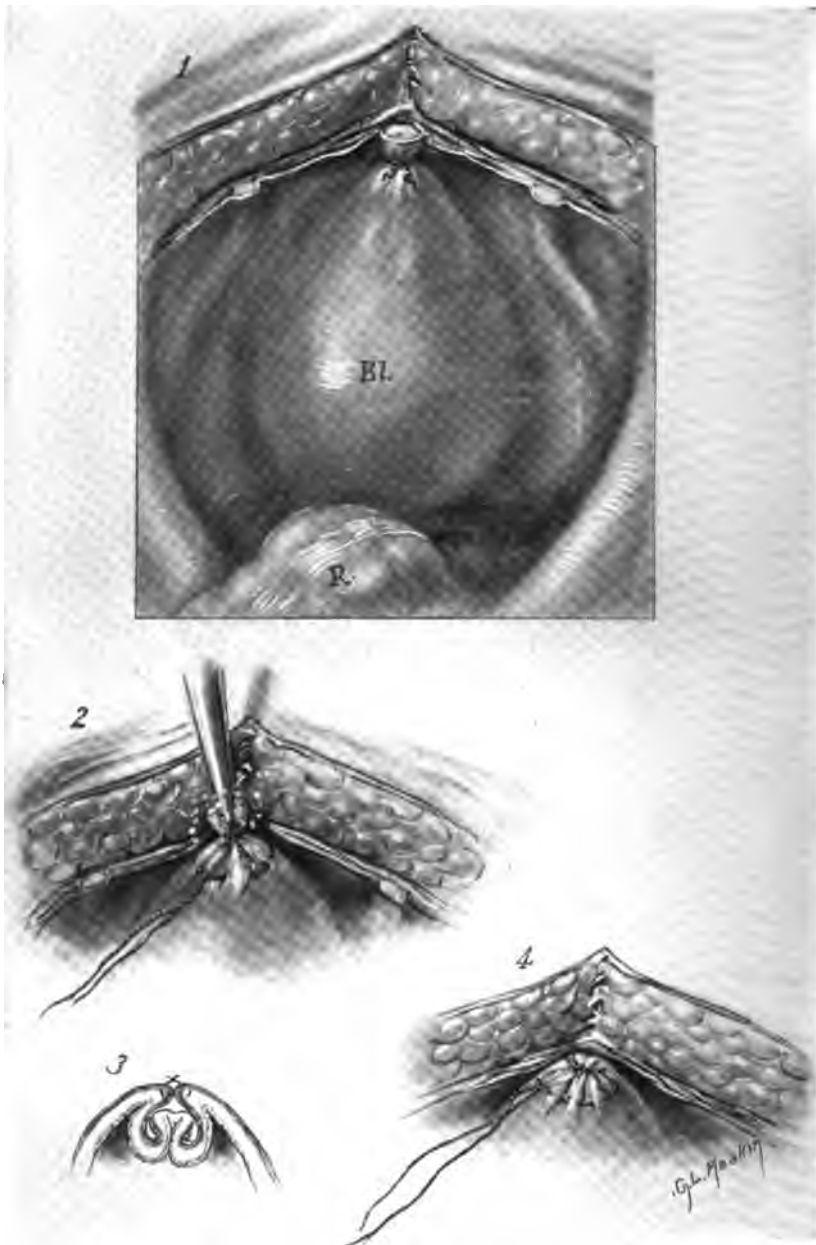


Diagram illustrating the development of the allantois.

FIG. 2.



- 1, Drawing showing the stump of the urachus ligated after the distal portion has been excised.  
2, Purse string applied after the urachus has been freed from the anterior wall of the abdomen.  
3, Stump has been inverted. 4, A second purse string is inserted beyond the first.

FIG. 3.



**Drawing showing the hernial sac opened revealing the fimbriated extremity of the tube and the ovary. The tube shows through the posterior sac wall.**



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forceps well down to where it begins to broaden out. A catgut ligature replaces the crushing forceps (Fig. 2 (1)). The remnant distal to the ligature is excised. The stump is next inverted by a purse-string suture (Fig. 2 (2), (3)). A second purse-string suture is applied to insure against leakage (Fig. 2 (4)).

The abdomen is now closed layer to layer and a small wick drain is inserted down to the fascia.

*Post-operative.*—Slight suppuration took place in the superficial layers, but cleared up in a few days. The patient left the hospital on the eighteenth day after operation.

Two weeks later he returned to the hospital and cystoscopic examination revealed a small puckering scar in the upper part of the bladder, the site of the invaginated stump of the urachus. The patient stated that he had been working every day and experienced no trouble referable to the bladder.

#### SLIDING HERNIA OF THE FALLOPIAN TUBE, WITH REPORT OF A CASE

The escape of either the Fallopian tube or ovary or the Fallopian tube accompanied by the ovary, through any of the usual hernial openings in the lower abdominal or abdomino-pelvic region is not particularly uncommon.

Heineck<sup>1</sup> has published a very interesting analytical review of some 137 undoubted cases which he was able to gather from the French, German and English medical literature from 1890 to 1910, inclusive.

A considerable number of cases have been reported since his review, and I am reporting this case with a view to placing it on record.

I have previously reported a case occurring in an infant of six months in whom a left-sided inguinal hernia of the congenital type became strangulated.<sup>2</sup> At operation the hernial sac was found to contain a gangrenous tube and ovary. The strangulation was evidently produced by torsion of the tube and ovary. The operative removal of the gangrenous structures and radical repair of the hernia after the method of Bassini was followed by an uneventful convalescence.

The present case is interesting in that the hernia is of the sliding type or *hernia par glissement* of the French writers. Moschcowitz<sup>3</sup> states that sliding hernias of the Fallopian tube are among the rarer

forms of true sliding hernias. Heineck refers to one case in which the sac was formed by the two layers of the broad ligament. In my case the anterior wall of the sac was made up of parietal peritoneum while the lateral and posterior walls were derived from the two spread-out layers of the broad ligament. The fimbriated extremity of the Fallopian tube opened within the lumen of the hernial sac while the remainder of the tube showed as a cord-like structure beneath the peritoneum, extending upward and backward toward the internal ring. Within the abdomen it could be distinguished as a sub-peritoneal cord-like structure passing over the pelvic brim and downward and inward over the iliac vessels to join the cornu of a very rudimentary uterus. No semblance of a broad ligament could be made out. The right tube, ovary and broad ligament were normal in their appearance. The uterus was scarcely larger than an almond.

REPORT OF CASE.—Miss M. K., aged ten years, was referred to me by Dr. Rabe of the Pædiatric Department of the Post-Graduate Medical School. The father informed me that the patient had for some time complained of pain in the left inguinal region. This pain was most marked after exertion and was accompanied by a tender swelling of the left labium majus. The father also stated that this same swelling had been noticed when the patient was a very young baby and was then most marked when the child cried.

*On examination* the child was an apparently healthy young girl with no evident malformation of external genitalia. A partially reducible left inguinal hernia was present. When asked to stand and increase the intra-abdominal pressure by coughing, a distinct enlargement appeared at the external inguinal opening and extended to the left labium majus.

*Operation* was performed October 16, 1919, under ether anaesthesia. The usual left oblique inguinal incision was made and the fibres of the external oblique divided. The hernial sac extended from the internal ring well down into the loose tissue of the left labium. The walls of the sac were thin but quite vascular, and there was very little sub-serous fat. There were no folds, adhesions, valvular or diaphragmatic constrictions present. The posterior wall of the sac was intimately adherent to the round ligament. The ovary was spread out over the posterior sac wall anterior to the fimbriated opening of the tube. The distal pole of the ovary had a fibro-muscular

cord extending to the fundus of the sac. The fimbriated extremity of the tube projected within the sac for a distance of 1.5 cm. The remainder of the tube could be followed, as a rounded cord beneath the peritoneum, to the internal ring and then within the abdomen to the cornu of the infantile uterus (see Fig. 3).

In order to do a radical repair of the hernia it was necessary to free the sac from the round ligament and then dissect the sac with its contained ovary and attached tube up to, and well within, the internal ring. The sac was then ligated and the excess removed.

Pathological report by Doctor Kirk showed that removed sac was made up of peritoneum, ovary and about 3 cm. of Fallopian tube.

A typical Bassini operation was done and the patient had an uneventful convalescence, leaving the hospital on the sixteenth day.

#### SUMMARY

1. This case presents a sliding hernia of the Fallopian tube through the left inguinal canal.
2. The contents of the hernial sac were the fimbriated extremity of the Fallopian tube and the ovary.
3. The hernia was partially reducible.
4. Operative ablation of part of the tube and the ovary was necessary to insure repair of the hernia.
5. The round ligament was preserved in its normal relation.

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## DIAGNOSTIC PROBLEMS IN ABDOMINAL SURGERY

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THE object in presenting this paper, is to emphasize the fact that the diagnosis and differential diagnosis of many abdominal and pelvic conditions and diseases, are not the accomplishment of the tyro in the field of medicine and surgery, and to stress the statement, with the assertion, that oftentimes the correct interpretation of abdominal and pelvic affections baffles the diagnostic skill of the experienced clinician in this particular field.

It is a well-known fact that pelvic and abdominal enlargements may be due to a multitude of causes and to attempt to name them all would be far beyond the limits of this brief paper. Among the pelvic conditions we enumerate: Cellulitis, peritonitis, hydro-hæmatopyosalpinx, cysts of the broad ligament, malpositions of the pelvic viscera, extra-uterine pregnancy, fibroid and fibrocystic tumors of the uterus, solid and cystic ovarian tumors. Among the abdominal tumors and enlargements we encounter, we enumerate: Pregnancy, hydatiform mole, fibroid or fibrocystic tumors of the uterus, ascites, enlarged spleen, encystic peritonitic effusion, cancer of the peritoneum, cyst of the broad ligament, omental cyst or solid tumor, hydronephrosis, renal cyst, pancreatic cyst, cyst of the mesentery, solid tumor of the abdominal wall, distended bladder, tympanites, phantom tumor, floating liver.

These tabulated lists, incomplete as they are, are merely subjoined to make the picture more complete and at the same time to illustrate the real difficulties that may be encountered, in certain instances, in arriving at a satisfactory diagnosis. The points which I wish especially to present, are best illustrated by the recital of the following cases, both occurring in young women, one four weeks after childbirth, the other in a single woman who had been regarded by her friends as being pregnant.

N. C., age eighteen, was admitted to the Philadelphia Polyclinic Hospital, June 6, 1920, with an abdominal enlargement, in which pain was not a prominent symptom. The woman was married and gave housework as her occupation.

She stated that she had always enjoyed the best of health, and had not even contracted the usual diseases of childhood. Her menstruation had begun at fifteen, it was regular in time, not profuse, nor did she suffer pain. She was married at seventeen, and a baby was born just twelve months later. Her family history offered nothing of interest. Father and mother and two brothers and sisters living and well. Two brothers died in infancy.

Her baby was born April 11, 1920. Her family physician, Dr. Percy H. Corson, of Plymouth Meeting, Pa., writes that at his first examination, there was great distention of the abdomen. The labor was a tedious one, for a shoulder presented and as the woman was a primipara, it became a difficult task to do a version.

After delivery and subsequently, the abdomen appeared abnormally enlarged. For three days after delivery she vomited and for a week she took no nourishment. Bowels were obstinately constipated.

One week after delivery, the visits of her physician ceased; but on the tenth day after delivery, she again sought his services because of a continuance of the abdominal distress. Two consultations were subsequently held, and on April 21, 1920, three weeks after the birth of the child, the patient came to the home of her mother in Philadelphia. Three weeks subsequently, or forty days after delivery, I was called in consultation by her attending physician, Dr. John Penza.

I found the woman in good physical condition; heart and lungs normal, normal temperature and pulse, also the blood normal. The urine was negative and there was no reaction to the von Pirquet test.

The abdomen was symmetrically enlarged from the pubes to the ensiform cartilage, and from flank to flank. Free fluid seemed to be present. The distress complained of was a feeling of distention and fullness, rather than of pain. Vaginal examination was negative, the cervix was freely movable and the fundus fairly well involuted.

A provisional diagnosis of abdominal ascites was made, and an exploratory incision was decided upon. Operation revealed the presence of a large, thin-walled multilocular cyst of the left ovary, which

weighed thirteen pounds (Frontispiece). The patient made an uninterrupted recovery and was discharged from the hospital June 30th.

The record of the other case which I now append, is in itself a corroboration of the oft-repeated statement that the ill of the human body do not always conform to the printed page of the text-book, but frequently present a sufficient number of cardinal signs and symptoms of other conditions and maladies, that may make a diagnosis difficult or impossible. The details of the case about to be here reported, offered a clinical picture resembling both an ovarian tumor and a pregnancy.

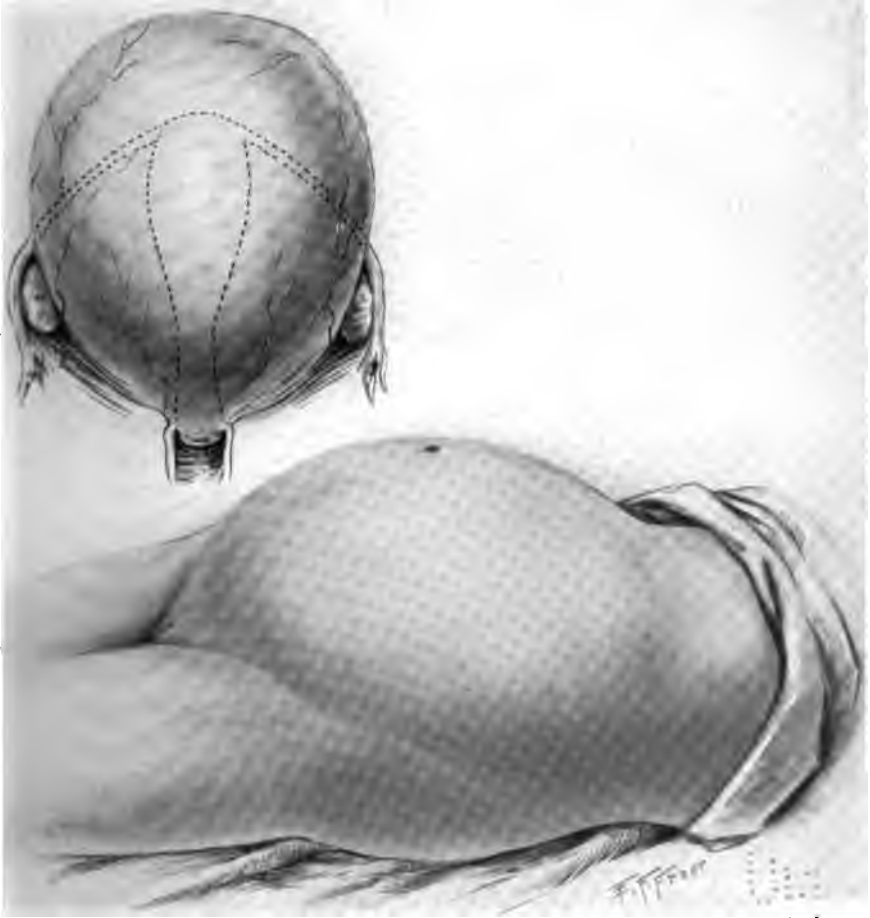
C. C., age thirty-six, weight 102, height 5 feet 2 inches, occupation hotel housekeeper, admitted to the Philadelphia Polyclinic Hospital, July 11, 1920. She presented the clinical picture of a woman in the eighth month of gestation. Her mother and father had died from pneumonia. There were nine other children; three brothers and two sisters are living and well; two brothers and two sisters died in infancy. The patient had measles and an attack of typhoid fever at twenty-one.

Six months before admission to the hospital, the patient noticed that her abdomen was enlarging and she stated that this increase in size was progressive. She had little pain, but rather a "distress" upon both sides of the abdomen. Three months before her entry into the hospital, she had suffered from vomiting—the vomiting bore no definite relation to the time of taking food; at times the ejected matter immediately followed the meal, at other times she vomited two or three hours after taking food. Her vomiting continued up until the time of her admission to the hospital. She suffered much pain in the legs—pains that were rheumatic in character, and from which she obtained relief by resting in the recumbent posture, as when retiring at night.

Her menstrual flow began when she was fifteen, was painless, regular and in every way normal. This continued throughout twenty years of her menstrual activity, or, up until three months ago, when the discharge became much more profuse, occurring bi-monthly; duration of the flow from a week to ten days.

Physical examination revealed an abdomen symmetrically enlarged in all directions, presenting the appearance of an abdomen near term. It was smooth and regular in outline and boggy in consistence. Percus-

FIG. 1.



Fibromyoma simulating pregnancy. Case No. 2.



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sion gave a solid, dull note. There was a line of pigmentation along the linea alba from the umbilicus to the pubes. The breasts were not enlarged, but showed a light areola around the nipples. A few drops of colostrum-like fluid could be expressed from the nipples and the vaginal mucous membrane exhibited a bluish discoloration. On the right side just below the level of the umbilicus was a hard mass, resembling a possible foetal part. Was this part of a foetus or was it a lobule of a multilocular ovarian cyst? At operation the enlargement proved to be a large fibrocystic tumor of the uterus, developing in the posterior wall (Fig. 1).

The operator was in doubt as to whether he was dealing with a pregnancy. On closer study it was found that the uterus had been pushed up by a fibrocystic growth, developing in the lower posterior wall. The distance between the cornua of the uterus was not much disturbed and in this way differed markedly from the pregnant uterus. In other respects the entire mass bore a close resemblance to the normal uterus at term. Hysterectomy by amputation was done. The tumor weighed six pounds.

These two cases illustrate some of the difficulties in the diagnosis of abdominal neoplasms.

Fibrocystic tumors of the uterus may resemble a pregnancy as well as ovarian cysts, so that even the most experienced surgeon at times will hesitate. In these instances the uterine cavity is commonly enlarged, the tumor forms one mass with the uterus, its consistency is softer than the normal uterus; hard masses are often to be felt in different parts of the tumor, and the patient often suffers from menorrhagia. The development is usually slower than ovarian cystoma and the constitutional symptoms are less marked.

# Medicine

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## A FATAL CASE OF ACUTE HEPATIC ATROPHY OF MEDICO-LEGAL INTEREST

By F. PARKES WEBER, M.A., M.D., F.R.C.P. (LOND.)

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A MAN (M. R.), aged twenty-nine years, came to the out-patient department of the London German Hospital (Dalston) on April 22, 1919, with a wound on the right side of his face. He was a demobilized Jewish soldier in the English army, and it transpired that on the preceding day in a coffee house in the East End of London he had been mixed up in a quarrel and an American (Russian-Jewish) soldier had stabbed him (right side of the face) with a pocket-knife. The patient refused to remain in the hospital, but he returned on the following day to have the wound dressed. He next came on April 26th, when he complained of more pain, and was admitted as an in-patient. On the morning of April 27th the wound was carefully examined under general anæsthesia and part of the blade of a pocket-knife was discovered and removed from the fractured malar bone. The operation lasted about twenty minutes and the anæsthetic used was chloroform. On April 29th slight jaundice was observed, and on the next day (April 30th) the patient seemed drowsy. The stupor increased, and there were two convulsive attacks affecting the left limbs. At 11 P.M. on the same day (April 30th), as there was a question of cerebral abscess on the right side of the brain, the skull was trephined in the right temporal region, but no abscess was found. This operation lasted about three-fourths of an hour, and chloroform was the anæsthetic used as far as any anæsthetic was required. The patient, who was already more or less comatose before the operation, remained comatose afterwards, but the jaundice gradually increased until his death, which occurred early on May 2nd.

At the *necropsy* the liver presented the typical macroscopic appearance of acute parenchymatous atrophy, as if the capsule was too big for the amount of hepatic glandular tissue which it contained. There was nothing else of importance to be noted excepting that

both the kidneys were fatty-looking. Microscopical examination of a piece of the liver showed extreme disintegration of the hepatic parenchyma and more or less degeneration or atrophy of nearly all the liver cells. There was practically no inflammatory reaction. Professor S. G. Shattock, who kindly examined a section with me, did not remember to have ever seen a more extreme example of hepatic atrophy. It has occurred to me even that the atrophic change in the liver went on increasing after the patient's death, and in this connection it should be noted that a certain amount of so-called "post-mortem emphysema" of the liver was likewise observed on microscopical examination. The accompanying *figure* is intended to illustrate one of the better preserved portions of the hepatic parenchyma under high magnification. The degenerated liver-cells have entirely lost their normal columnar arrangement.

In this case the increasing jaundice and coma constituted the typical clinical picture of "icterus gravis," and the condition of the liver and kidneys found at the post-mortem examination suggested that the case was one of so-called "late chloroform poisoning" in a man who was, for some unknown reason, specially predisposed. No sepsis nor fever had been present to suggest that the condition of the liver could be due to infection. There was no evidence of recent syphilis.<sup>1</sup> There was nothing in the history of the case to suggest any possible poisoning by phosphorus, trinitro-toluene, salvarsan, or other substance acting in more or less the same way on the liver.

One consideration, however, seems to me worthy of mention, and that is that the degree of disintegration of the hepatic parenchyma was almost greater than one would expect in any case of so-called late chloroform poisoning. Although at the inquest the jury returned a verdict of manslaughter against the American soldier, the latter was afterwards dismissed at the Police Court; the judge held, I understand, that the acute atrophy of the liver might have occurred independently of the chloroform and independently of the wound. For permission to describe this case I am indebted to my surgical colleague, Mr. A. Compton.<sup>2</sup>

On the whole, it must now be admitted, I think, that the icterus

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<sup>1</sup> Cf. F. P. Weber, "Acute Hepatic Atrophy in Early Syphilis," *Proc. Roy. Soc. Med., Pathological Section*, London, 1909, Vol. ii, pp. 113-122.

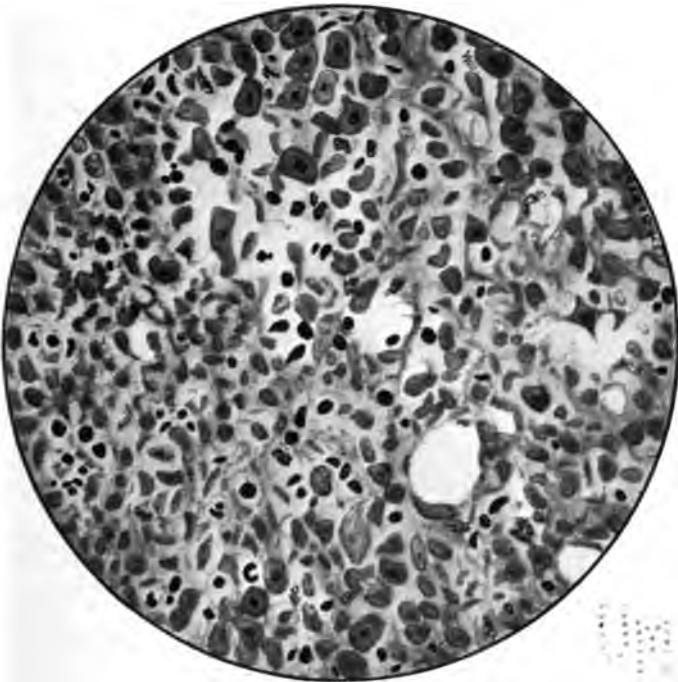
<sup>2</sup> The case was not actually under his personal treatment.

gravis and hepatic atrophy were probably manifestations of so-called "late chloroform poisoning," and the man would probably have died even had the second operation not been performed.

This reminds me that in certain cases, when there is supposed to be something wrong with the liver and when cholelithiasis is suspected, an exploratory operation is not unlikely to be made. There is, ordinarily, a chance of the surgeon's doing good and relieving the symptoms and very little chance, with the present knowledge and excellent surgical technic, of any harm coming of it. Nevertheless, if the symptoms in question happen to be due to commencing sub-acute atrophy of the liver or are premonitory symptoms of such a condition, then an exploratory operation under chloroform anæsthesia may well cause grave and even fatal mischief. At the laparotomy nothing obvious would be found, and the operator would consider it his duty not to close up the abdomen without having made a thorough and exhaustive search for any possible "surgical" cause of the symptoms in question. The operation is therefore likely to be prolonged, and the chloroform is thus given an extra chance of exerting its deadly influence on the already predisposed parenchyma of the liver.

It seems to me that the case of a man in whom my father was much interested was an example in point. The patient, born in 1866, had suffered from dysentery in South Africa about 1900, and after that he had become subject to recurrent "bilious-like" attacks. When seen during one such attack he was slightly jaundiced, but the fæces were not colorless and the urine did not contain bilirubin. My father and a London surgeon thought that the symptoms did not constitute a reason for any operative interference. In 1906, however, the patient had another attack of jaundice; an exploratory operation was performed (by an excellent surgeon at Dublin), and he died. At the operation no gall-stones were found, and there was no evidence of any obstruction to the flow of bile in the bile-ducts. The post-mortem examination was reported by Professor A. C. O'Sullivan at the Pathological Section of the Royal Academy of Medicine in Ireland, on May 4, 1906. The liver weighed sixty-nine ounces, was larger than normal, of a greenish-yellow color, and the capsule was smooth. On section one could see greenish-yellow areas, separated by lines of red material. Microscopic examination showed that the centre of the

**FIG. 1.**



**Section from the liver in the case of M. R. The degenerated liver cells have entirely lost their normal columnar arrangement.**

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lobules was degenerated (so-called centro-acinous degeneration or centro-acinous necrosis of the liver), and a zone of comparatively healthy liver cells was to be seen towards the periphery. The degenerated cells in the centre showed an almost entire absence of protoplasm, and their nuclei were shrunken and deformed. A great many cells were filled with granules of bile-pigment, and the bile-capillaries were filled with plugs of inspissated bile. There was no inflammation of the smaller bile-ducts, nor was there any sign of increased connective-tissue formation. The cells were separated from each other and had lost their columnar arrangement. No bacteria were found.<sup>3</sup> It should be here noted that a centro-acinous necrosis of the liver can be experimentally produced by chloroform anaesthesia in dogs.<sup>4</sup>

With the preceding case one may compare "A Case of Subacute Atrophy of the Liver," recorded by S. G. Scott in 1905.<sup>5</sup> There were uncertain symptoms for a year or two, followed by an attack of jaundice, and the possibility of cholelithiasis was considered. At the exploratory operation no cholelithiasis was discovered, and the patient (a woman, aged twenty-three years) died from hemorrhage on the following day. The necropsy proved the case to be one of subacute hepatic atrophy. Clearly in that case subacute hepatic atrophy preceded the operation and the anaesthesia.

The following case from St. Bartholomew's Hospital, London, was recorded by G. L. Keynes in 1914.<sup>6</sup> The patient was a young woman, aged twenty-four years, who had given birth to a healthy child on June 13, 1913. Shortly afterwards she noticed that she was becoming jaundiced, and she suffered from loss of appetite and vomiting. In September, 1913, she had severe pain in the right hypochondrium, accompanied by rigors, sweating and vomiting. In December, 1913, there was another, but less severe, attack of pain, and after the attack she became more deeply jaundiced. She had a third attack of pain in January, 1914, and the jaundice again became

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<sup>3</sup>A. C. O'Sullivan, "Usual Degeneration of the Liver," *Transactions of the Royal Academy of Medicine in Ireland*, Dublin, 1906, Vol. 24, p. 441.

<sup>4</sup>G. H. Whipple and J. A. Sperry, "Chloroform Poisoning," *Johns Hopkins Hospital Bulletin*, Baltimore, 1919, Vol. xx, p. 278.

<sup>5</sup>S. G. Scott, "A Case of Subacute Atrophy of the Liver," *Transactions of the Pathological Society of London*, 1905, Vol. 56, p. 298.

<sup>6</sup>G. L. Keynes, *St. Bartholomew's Hospital Journal*, London, 1914, Vol. 21, p. 147.



deeper. After this the jaundice varied in intensity. In March, 1914, she was admitted to the hospital, and a provisional diagnosis of cholelithiasis was made. This was not confirmed by an exploratory operation. The gall-bladder had thickened walls and was excised; a subsequent microscopical examination showed it to be the seat of acute catarrhal inflammation. After the operation the patient appeared to be definitely less jaundiced, and the wound healed by first intention. On the ninth day after the operation, however, she was obviously worse, and there was hæmatemesia. Death occurred on the following day, April 9, 1914, with signs of acute anæmia. Post-mortem examination of the abdomen showed the stomach, small intestines and peritoneal cavity full of blood, the bleeding having apparently proceeded as a general oozing from the omentum and mucous membrane of the stomach and intestines. The liver was rather small and on section was found to be studded with enormous numbers of small, brilliantly yellow areas. Microscopical examination showed degenerating liver cells and strands of fibrous tissue enclosing numerous so-called "regenerating bile-ducts." The case was one of icterus gravis simulating cholelithiasis. Obviously, in this case also, the pathological condition of the liver found at the post-mortem examination must in large part have existed before the operation.

Steinheil,<sup>7</sup> in 1911, described the case of a deeply jaundiced woman, aged thirty-nine years, who had her gall-bladder removed on account of gall-stones. Chloroform and ether were used in the anæsthesia. She died with anuria three days after the operation. Steinheil thought that the anuria in the case in question depended on a condition existing before the operation was performed.

Clairmont and von Haberer<sup>8</sup> (in von Eiselsberg's Clinic at Vienna, 1910) had already described cases of cholelithiasis and biliary obstruction in which operation under general anæsthesia was followed by oliguria or anuria and death.

In regard to the dangers of chloroform anæsthesia in jaundiced patients, French communications in 1910 by Tuffier,<sup>9</sup> Sieur,<sup>10</sup> and

<sup>7</sup> Steinheil, "Ueber Icterus gravis und Anurie," *Beiträge zur klin. Chirurgie*, Tübingen, 1911, Vol. 76, p. 629.

<sup>8</sup> P. Clairmont and H. von Haberer, "Ueber Anurie nach Gallensteinoperationen," *Mitteil. aus den Grenzgebieten d. Med. u. Chir.*, Jena, 1911, Vol. 22, p. 159.

<sup>9</sup> Tuffier, "Accidents chloroformiques cher les ictériques," *Bull. et Mem. de la Soc. de Chirurgie*, Paris, 1910, Vol. 36, p. 488.

<sup>10</sup> Sieur, "Sur les ictères post-chloroformiques," *ibid.*, p. 519.

Quénu<sup>11</sup> before the Paris Society of Surgery, may also be referred to. Tuffier quoted two cases recorded by Foa (in Italy in 1907) in which icterus gravis supervened after chloroform anæsthesia in patients with latent hepatic cirrhosis; the presence of the cirrhosis was verified in both cases by microscopic examination after the necropsy. In England the late Leonard Guthrie, from 1894 onwards, repeatedly drew attention to the fact that there is a state of the liver of some kind or a constitutional state that predisposes the patient to hepatic atrophy and renders chloroform anæsthesia especially dangerous.<sup>12</sup>

*Experientia docet!* But the life of a doctor or surgeon ought not only to be peculiarly rich in experience, but would have to be prolonged by at least a hundred years if he is to avoid all the traps and snares which the symptoms of disease set for him. Although in many recorded cases the chloroform anæsthesia may not have been the chief or only cause of death, and a condition of icterus gravis with subacute hepatic atrophy may have been present before the operation, yet in some cases the chloroform anæsthesia was probably the main cause of death.

Doubtless an exploratory operation under chloroform anæsthesia has occasionally been performed when an early stage of hepatic atrophy, or a predisposing condition, was already present. In regard to "traps" of this kind, I would refer to the possible occurrence of the rare granulomatous form of iodide eruption in a patient under treatment by potassium iodide for real or supposed syphilis. The iodide granulomata in such cases may perhaps be mistaken for syphilitic lesions, with the result that the real cause of the condition (the iodide) is continued or even increased in its harmful action.

#### DESCRIPTION OF ILLUSTRATION

Section from the liver in the case of M. R. One of the better preserved portions of the hepatic parenchyma under high magnification. The degenerated liver-cells have entirely lost their normal columnar arrangement.

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<sup>11</sup> E. Quénu, "Du danger spécial du chloroforme chez les icteriques," *ibid.*, p. 629.

<sup>12</sup> Cf. Leonard Guthrie, "On the Fatal Effects of Chloroform on Children suffering from a Peculiar Condition of Fatty Liver," *Lancet*, London, 1903, Vol. ii, p. 10; also many other shorter contributions by Guthrie.

## A LECTURE ON TUBERCULOSIS\*

By PROFESSOR CLEMENS FREIHERR VON PIRQUET

Vienna.

THE infection with tuberculosis is almost always brought about by breathing tubercle bacilli into the lungs. The microorganisms settle at first in the bronchi and there lead to a small area of infection (Ghon's area). Thence the infection of the regional lymphatic glands results by way of the lymph passage into the mediastinum, principally in the region of the bifurcation of the trachea. The other entrance portals of the infection occur much more rarely than the bronchogenic form mentioned. The placentagenic infection of the foetus occurs only exceptionally and almost always leads to death in the first few months of life. The stomatogenic infection of the oral mucous membrane and the dermatogenic infection from any part of the outer skin are likewise rare exceptions. The infections on the prepuce at circumcision belong to the dermatogenic kind. The enterogenic infection from reception of tubercle bacilli into the intestinal canal plays a greater part. On the Continent, where it is customary to give the milk to infants only when boiled, it is rare; on the other hand, it appears to occur frequently in England and America, where cow's milk is preferably given raw. In all these kinds of infection the so-called primary stage consists in the entrance portal and in the regional lymphatic glands; the pathologic findings are different only in the placentogenic tuberculosis. This primary stage is followed by the secondary one, in which the tubercle bacilli are carried into the circulation from the primary area, in particular from the tuberculous lymphatic glands, and cause metastases on the most various parts of the body. We see the worst forms after the development of numerous small areas in the cerebral meninges, which under the picture of tuberculous meningitis leads to certain death. The miliary tuberculosis of the lungs is not so absolutely fatal. The miliary tuberculosis of the skin with the various clinical pictures of tuberculosis is comparatively harmless; of late years we have learned to know this form better. Isolated tubercles in the bones, in the joints, in the

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\* English by B. Lewis, Wein xix, Vegagasse 15.

subcutaneous tissue, in the brain, in the testicle, etc., and their subsequent phenomena in the glands are generally familiar. Finally, we have to mention as of this stage phlyctenula of the eyes, scrofulous catarrhs and the swelling of nose and lips which Schick brings into connection with the accompanying excretion of the tubercle bacilli through the mucous membranes.

The so-called third stage presents an entirely new picture. According to our present opinion, it is based on another kind of reaction of the organism after puberty, while during childhood the primary areas in the lungs may gain in extent at the most various spots. We see with, and during, puberty, and preferably in the upper parts of the lungs, processes inclining to cavernous formations which are distinguished from the infantile processes by a chronic torpid course. In the scrofulous skins of children we find similar differences from the lupus of adults or from the chronic larynx phthisis in later years.

In all this we recognize that the cause of infection is to be distinguished from that of disposition. It seems that every person is susceptible of infection, that there is no absolute immunity from tuberculosis. As a matter of fact, there are in the large European cities but few individuals older than twenty years who have not had an infection.

However, the differences in the clinical picture result from the various dispositions. Herein the disposition of a family or of a people seems to play a smaller part than the age disposition. While in the new-born child the disease never ends with the primary stage alone, but always develops secondary symptoms of which the child mostly dies, the healthy six-year-old child almost always throws off its tuberculosis without secondary phenomena. Probably at later age the temporary disposition of sub-nourishment acts on the tuberculous process. It appears that the tubercle bacilli remain alive for years in the regional lymphatic glands; they are only imprisoned there, not destroyed. When the organism is enfeebled by sub-nourishment they are able to break through the bars, to get into the circulation and, after puberty, to form metastases in the most various organs of the body, principally in the lungs. The organism undergoes the most important change of disposition owing to sub-nourishment. This experience regarding tuberculosis was very extensively gained during the war. According to the investigations of Wassermann, Jr., the line

of deaths from tuberculosis in Prussia runs parallel with the decrease of food rations after the interval of about one year. Analogous observations—although not in such precise manner—were made in Austria also. Every long period of sub-nourishment in an organism infected with tuberculosis can lead to its spread, whether proceeding from lack of food or of appetite. Moreover, the tuberculous affection itself has almost a specific effect on the appetite; that is, as the tuberculosis increases the patient grows more and more particular as to what he eats; a vicious circle arises which ends with the complete emaciation of the patient while the disease gains more and more ground.

We can make the *diagnosis of tuberculosis*, firstly, by the establishment of various clinical forms of tuberculous affections, such as spondylitis fungus and lupus; secondly, by the demonstration of tubercle bacilli, and thirdly, by the establishment of the specific antibodies in the tuberculin reaction. In adults this tuberculin reaction is of less significance than in children, as it only means the proof of an infection that had taken place, not the presence of a tuberculous affection or the continuation of the process. The younger the children are the more importance must be attached to the infection as such. We therefore perform the reaction on every child at the dispensary whom we suspect to be tuberculous, and in the hospital on every child.

The test is performed on the outer side of the right lower arm in the following manner: The skin is washed with ether, then two small drops of undiluted Koch's old tuberculin are put on at a distance of about ten centimetres from each other; then with a special lancet a drill is made at first midway between the two drops and then within each drop; after forty-eight hours the reaction is tested. If negative, the Mantoux test may follow, which consists in an intracutaneous injection of one-tenth cubic centimetre of a one per cent. solution of old tuberculin.

In my clinic the *treatment of tuberculosis* consists essentially of a definite feeding regulation. This is based on the thought that tuberculosis can only be cured by the antibodies which must be formed in the organism itself. These antibodies are formed the better and more plentifully the more intense the entire metabolism is and the more excess of metabolism remains for the antibody formation. In my opinion, all the cures which in the course of time have been

FIG. 1.



A favorite patient.

FIG. 2.



Lad seventeen years old, see p. 64.

FIG. 3.



Roof garden.

FIG. 4.



Children suffering from tuberculosis.

stated for tuberculosis culminate in the therapy of feeding. The patients are sent now to the seashore, then to the mountains, now in the desert, then into warm valley; all these resorts have in common only the circumstance that the patients there find good care and, under the impression of change of scene and freedom from home cares, begin to eat. The rest cure effects a saving of expenditure and allows of a greater excess of metabolism, while being out in the sun and exposure of the skin out of doors stimulate the appetite. The change of place and the numerous psychic stimulants may be dispensed with if a definite amount of food be prescribed and if the patients be ordered to take an open-air cure at home combined with suitable rest intervals. The large roof station for one hundred tuberculous children in the centre of Vienna, a city so notorious for tuberculosis, furnishes the proof of my assertion. On an average, the children remain there half a year. They are in the open air summer and winter, night and day, being in the daytime clad to suit temperature and well covered at night. The children thus become so accustomed to living in the open air that in summer they wear only short drawers, and it is only during cold winter nights that the little ones must be provided with a hot-water bottle. The children are indoors only while washing and bathing, at meals and at school, in all about four hours of the twenty-four. In summer, school is also partly kept in the open air. The most important function of the day is eating, for *food is the medicine for tuberculosis*. The children receive five meals at intervals of three hours: three principal meals, morning, noon and evening, with two slighter ones in between. These slighter ones are the same for all, but the principal meals are graded according to the various *food classes*. The food class is determined in accordance with the sitting-height. In the lowest the children receive the nutritive value of two litres of milk daily (two kilonems); in the highest class, of four and one-half litres (four and five-tenths kilonems). This, however, means the nutritive value only, for milk itself constitutes only a trifling part of the nutrition. We give meat only one to three times a week and to give eggs we are at present unfortunately altogether unable. The chief part of the food consists in flour, fat, potatoes and vegetables, a diet which does not materially differ from that which they can receive under normal conditions. The results of this nutrition and open-air treatment are very sat-



isfactory. At first we subjected to the open-air treatment only the cases of tuberculosis of the bones, of the joints, of the glands and of the skin. Since, however, the results in such cases were so favorable we went a step further and took up on the roof the cases of tuberculosis during the first years of puberty. This tertiary stage, at the age of twelve to fourteen years, we heretofore regarded as almost hopeless, but have now seen that a satisfactory number of cases come to a cure. We are particularly pleased about a lad of seventeen years who has now been living on the roof for three years, goes to school from there and at the same time acts as gardener of the roof, and because he is so good we have exceptionally permitted him to remain in the children's hospital beyond the prescribed age. His cell consists in a sort of tent in a corner of the roof and is furnished in the most primitive manner, merely with a narrow cot covered with a woolen blanket, a small table to hold his books and a receptacle for his garments and trifling treasures, leaving just room enough for him to move about. On waking in the morning—after a healthy night's sleep—he has the sky above him and the wonderful panorama of the city below with its encircling hills and the broad glittering ribbons of the Danube before his eyes. This fortunate lad has gained twenty kilograms in weight since being with us, and the bilateral apex tuberculosis, combined with severe bacillary sputum, is completely healed, and he is the picture of health.

But we have the greatest pride in a case of tumor of the cerebellum which was established as tuberculous, in so far as such a diagnosis can be established on the living body. The patient suffered from severe epileptic attacks and symptoms of cerebral pressure, and an operation had been proposed by Professor Marburg. The treatment, which lasted at the clinic from January to November, 1918, and was then continued at home by his mother, resulted in a perfect cure; at present, 1920, he is healthy and vigorous and perfectly normal mentally. There are two other similar cases of cerebral tumors which have been completely cured. We cannot understand why the tuberculous process which, as we know from experience, can be cured in all other organs of the body, should not be curable if localized in the brain, only that in these cases the question of nutrition is particularly difficult. Complete absence of appetite and the irritation to vomit are the

cause that definite, quantitative directions as to nutrition cannot be carried out.

Tuberculosis is the severest infectious disease of childhood. In view of the extraordinary spread of the tubercle bacillus among the human race it will probably for some time be impossible to bring up children free from tuberculosis and keep them so. Our first efforts must be directed to keep infants free from infection. By enlightening all parents as to the fact that at this age infection is particularly dangerous, and that consumptive persons, indeed all persons who have a cough, should not be permitted to come near the infants. At a later age the danger of infection is not so great; that is, if we succeed in avoiding sub-nourishment. Sub-nourishment means danger of the tuberculosis spreading in every infected person. When such spread has come to pass, the most important thing is a treatment by quantitative nutrition, the appetite to be encouraged by plenty of sunshine and fresh air and the waste of body diminished by a regular mode of life.

When these three general principles shall be recognized all the world over, we shall succeed in gradually restricting this plague of humanity and in reducing the percentage of mortality from tuberculosis to a minimum degree.

## VACCINIFORM HYDROA

By PAUL BONNECAZE, M.D.,  
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It is a rather astonishing fact that even to-day there still reigns some misunderstanding on the subject of vacciniform hydroa. Before entering upon an explanation of this point I would first examine what lesion should be understood by summer eruption and the cutaneous lesion under consideration, which have been erroneously looked upon as identical by a large number of dermatologists.

In 1888, Hutchinson gave the name of summer eruption to a typical case of vacciniform hydroa, whose real nature he did not appreciate and which he regarded as a peculiar variety of xeroderma pigmentosum, with which lesions it has no connection. The eruption described by Hutchinson occurred in a child, recurred each year during hot weather and was seated on the exposed portions of the cutaneous surface. The lesions occurred in the form of vesicles and after they had subsided left cicatrices. Since reporting this case the regretted London surgeon described under the same name a certain number of eruptions offering similar characters, but differing by one point, namely, the absence of cicatricial formation.

Thanks to Crocker's writings in particular, we are at present familiar with a group of dermatoses which can very properly be termed recurring summer eruption and possessing as a common bond of union an identical etiology. Their onset is in childhood, they recur each year in the spring or summer, are seated on the exposed portions of the cutaneous surface, but differ among themselves by the very characters of the eruption, which may be erythematous, papular, erythematopapular, vesicular, pustular, etc., but none of them ever result in cicatricial formation after recovery.

By its etiological characters, vacciniform hydroa should be placed under the heading of recurring summer eruptions, but the very nature of this eruption separates it from the former lesions in a most absolute manner; the lesions of vacciniform hydroa are invariably vesicular or bullate and *leave behind permanent cicatrices*.

The mistake of Hutchinson and other writers who imitated him was to identify two terms which are not synonymous. The denomination of summer eruption is, for that matter, much too general and too comprehensive, a fact that explains why cases of dyshidrosis and even simple lesions of insolation have been classified under this heading.

It is, therefore, proper to retain the name of vacciniform hydroa for the affection first described by Bazin, or as Crocker would have it, estival vacciniform hydroa, which possesses the advantage of indicating the principal character of the eruption, which is to leave vacciniform cicatrices behind, and also to recall one of its etiological peculiarities.

I therefore propose to reserve the term of summer eruption or, better still, that of recurring summer eruption, for all recurring eruptions of hot weather and which *do not* leave cicatrices.

One fact dominates the etiology of vacciniform hydroa, namely, the influence of atmospheric agents and, above all, solar rays. Their act is made evident in all the recorded instances of the affection.

In the majority of cases the eruption occurs shortly after the patient has been exposed to the sun. Exceptionally it has been known to occur without any exposure to solar rays, merely from the influence of a sharp wind or intense heat. In a case reported by Graham the history of the patient is most interesting in this respect. A child of six years of age, whose lesions were ordinarily the result of exposure to the sun, suffered an outbreak of the eruption after exposure to a sharp draught of hot air which suddenly burst through the window, the patient having been kept in his room on account of an inter-current disease.

Various data appear to prove the direct and irritating action of the solar rays in the production of the lesions. Thus the eruption is more frequent in the spring and summer, the luminous and caloric intensity of the rays being at their maximum at these seasons of the year. Secondly, the parts involved are precisely those which are ordinarily or exceptionally exposed to the action of atmospheric agents and unprotected by the clothing.

The sites of predilection for vacciniform hydroa are the face, particularly the nose and ears, then come the cheeks, the dorsal aspect of the hands, and lastly, the neck, legs, and feet when bare.

In some cases the eruption has become generalized over the back.

the arms, and gluteal region, but in these circumstances the elements composing the eruption are always discrete, developing, as they do, on abnormal sites. Exceptionally the eruption has been known to appear on the mucous membranes; Bazin met with it on the buccal mucosa, Constantin on the lips and tongue, Moller on the conjunctiva, Ledermann and Halberstaedter on the cornea.

The eruption occurs very rarely in winter, and in those rare instances where it has developed the patients had been exposed to the solar rays with snow. I am aware of only two cases (these have been reported by White) where the influence of solar rays appear to be about *nil*.

The eruption is eminently recurring in type, a yearly recurrence being the rule. The first attack is always met with in childhood, any time during the first to the tenth or twelfth year. The maximum of frequency appears to be during the first or second year of life.

Boys appear to be more prone to the affection in the ratio of 7 to 3. The process is more common in England and Germany, somewhat less so in America, and is rather rare in France. The pigmentation of the skin in certain colored races does not seem to prevent them from having vacciniiform hydroa, because I. Moreira observed a case in a little colored girl, whose father was a mulatto and the mother white.

From puberty on the attacks decrease in frequency and in intensity, and finally cease to recur between the ages of twenty to thirty years. There are, however, exceptions to this rule, as Parish met with it in a patient thirty-three years old and Lindser's patient was forty-four years of age; but such instances must be extremely rare.

The disease is neither hereditary nor familial. The eruption of vacciniiform hydroa when occurring in several members of the same family is simply a coincidence. The following hypothesis has been upheld for some years past for explaining the relative rarity of vacciniiform hydroa as compared with the extreme frequency of its causative factors, namely, that in certain subjects a susceptibility, a peculiar irritability of the skin to atmospheric agents, particularly by light, comparable to the idiosyncrasy of hereditary bullate epidermolysis, with this difference, that the predisposition of the skin in vacciniiform hydroa is congenital, but not hereditary.

From what has been said above facts are evident but explain nothing. During the past few years an attempt has been made to in-

crease our knowledge of the nature of hydroa vacciniiform and a number of researches have been undertaken in this direction. To these I will briefly refer.

Finsen experimented with the action of light on the healthy skin, that is to say, a skin not susceptible to the action of light. Covering the integuments of the arm with a thick layer of black paint, he exposed the limb thus prepared to the rays of an intense sun. After three hours of insolation he noted the following changes: In the first place, over the part protected by the paint nothing was observed after the protective layer had been removed. Over the unprotected parts, on the contrary, an intense erythema occurred, but only some hours later, and disappeared after a certain time, leaving behind a marked pigmentation.

In a second experiment, Finsen again exposed the same arm to the sun, but this time without the paint. He noted that the parts which were pigmented by the previous insolation had remained the same, while the neighboring non-pigmented skin alone presented an erythema. From these experiments Finsen concluded that the prolonged action of light provokes an erythema on the healthy skin and later pigmentation, which becomes a means of defense of the skin against the irritating influence of light.

Unna, Widmark and Moller have also studied by means of similar experiments the action of light on hydroa vacciniiform and obtained variable results extending from erythema to a vesicular eruption.

More recently Ehrmann, resorting to these experiments, was able to produce the bullous lesions or even the pustules with the necrotic process of certain of their elements which resulted in cicatricial formation. Besides experimenting with various spectral radiations, Ehrmann noted that the irritating action of light on the skin, which was *nil* for red radiations, became very marked for blue, still more so for violet and attained its maximum with the ultra-violet rays. Therefore, it is the rays with a short wave and chemical rays that possess the maximum of irritating power. In all these experiments the calorific rays of the spectrum were carefully filtered and eliminated, so that their action cannot be incriminated in the production of the lesions observed.

These results are not contradictory; they merely show that light is an irritant for the skin, that the latter reacts in various ways,

according to its particular degree of irritability and in a lesser degree according to the quality and intensity of the luminous action.

This appreciation is in complete accord with clinical facts. We know, in fact, that there exists an entire scale of cutaneous affections which are undoubtedly provoked by the action of light.

These dermatoses are characterized by erythematous, papular, vesicular and pustular eruptions which are recovered from without leaving any trace other than a transitory pigmentation. These are *the recurring summer eruptions*.

At their side is placed hydroa vacciniiform, characterized by a vesicular or bullous eruption of a necrotic type and invariably resulting in scar tissue formation of a permanent kind.

Therefore, we are led to assume that hydroa vacciniiform is not a malady *per se*, but simply a syndrome—one of the manners of reaction of the skin to light and the most serious of all.

As to the intimate nature of hydroa vacciniiform we are as yet unable to understand it. I. E. Graham has invoked a vasomotor reflex action induced by light. Leredde, taking into consideration the eosinophilia, noted in some few instances, likens hydroa vacciniiform to Dühring's disease and is inclined to regard the eruption as the effect of a toxidermia; but this theory is not admissible.

It is preferable to place hydroa vacciniiform among the reactions which arise in congenitally predisposed skins. While some epidermes engender bullæ from the influence of trauma (*epidermolysis bullosa*), the hydroa of Bazin produces them under the influence of light. The bulla is in this case a reaction—a singular one, it is true—in the same way that erythema, dyshidrosis or other varieties of estival eruptions produced by light and heat are.

To Bowen, of Boston, is due the credit of the first histological studies on hydroa vacciniiform. On the already umbilicated vesicles he found the following conditions: In the first place, a necrosis of the deep cells of the epidermis and the superficial and middle layers of the chorion. The superficial strata of epidermis and the horny layer were intact. The tissues surrounding the necrotic area were little involved and simply infiltrated. No bacteria could be discovered in the preparations.

Mibelli has described as initial lesion a primary diffuse dermatitis and an intense fibrino-leucocytic exudate resulting in the formation

of vesicles which form in the middle layer of Malpighi's mucous body. These vesicles are formed, not by a degenerative change in the cells of Malpighi, but by a dilatation of the intercellular spaces and the pushing aside of the cells.

Later on, the persistence of the inflammatory phenomena produces a necrobiotic process which destroys a more or less extensive area of the papillary bodies and ultimate repair by cicatrization.

Eddowes gave a description much like that of Mibelli and insisted on the influence of the cedema in the genesis of the necrotic process, and as treatment proposed early incision of the eruptive elements in order, if possible, to avoid cicatricial formation.

Moller has described a process of vesiculation quite similar to that of Bowen and Mibelli. He also explains the umbilication by the facts that the serum of the bullæ progressively transudes at the surface of the element and as the intravesicular pressure thus becomes diminished, the vesicle falls in at its centre. The writer also observed numerous small hemorrhages in the papillary bodies.

Malinowski states that he has noted lesions identical to those already described and considers the necrosis of the epidermis as an initial lesion and which later extends to the dermis.

Constantin examined histologically a young bulla before umbilication had taken place. The bulla was seated between the epidermis which appeared to have been raised up *en masse*, while the chorion was filled by a fibrino-leucocytic exudate, its single cavity not showing any partitions. In its *ensemble* the dermis offered throughout the sections signs of inflammation, made evident particularly by rather dense perivascular collars; there were few inflammatory cells infiltrated between the fasciculi of the deep dermis. On the contrary, the papillary body throughout its extent presented a rather marked diffuse inflammatory infiltration.

Around the bulla the epidermis appeared generally normal. At several spots, however, were seen, at its limits with the papillary body, islands recalling the spongoid state described in eczema and which appeared to prepare the formation of the bullæ. At these spots the cells "*en pallissade*" and the deepest Malpighian cells were violently dissociated, spread apart from each other and leaving between them lacunæ into which some leucocytes had emigrated. The disturbed cells had become profoundly involved, the majority could no longer



be recognized; they were granular and affected the most varied shapes, while some had been completely destroyed, as was evident from the great amount of protoplasmic detritus and nuclei undergoing pyknosis.

The bulla was constructed as follows: Its floor was represented by the papillary body, which was infiltrated and limited on the side of the cavity by a festooned line. Its vault was formed by all the subsisting epidermic layers, which were raised up *en masse*. These layers differed from each other at the centre and on the sides of the bulla: At the centre, the epidermis was very thin on account of the partial destruction of the deepest layers and accessorially from the pressure to which it was submitted; its cells were greatly flattened and their nuclei did not stain or only slightly. On the sides of the bulla the epidermic cells were, on the contrary, greatly increased in size, their protoplasm was very clear, and their look recalled that of elder-pith when seen under the microscope.

This is what Audry and Dalous have called medullation change and which, for that matter, offers nothing special at this area because it is found quite frequently in the neighborhood of the bullæ. Externally, the bulla was covered by a normal horny layer adherent to the sides, but at the apex it was thin and exfoliating.

In the cavity the epidermis was limited by a layer of dissociated cells on which the fibrinous network was inserted. The cavity of the bulla, which was single, was filled by a stroma of fibrin with very fine, close meshes. This network was inserted on the dissociated deep-seated Malpighian cells on the sides and dome, but it was not in contact with the dermic floor, from which it was separated by a layer of liquid which had coagulated and contained some leucocytes. The fibrinous reticulum contained, for the most part, white polynuclears within its meshes, and these were very much more numerous at the periphery.

Finally, nowhere in the dermis could foci of necrosis be discovered, but it is to be remembered that this histologic examination was made on a young bulla.

Hæmatologic and urologic researches are as yet too few to be able to give any general data. The blood of patients with hydroa vaccini-form which has been studied by a few writers has given somewhat dissimilar results. In two cases White found a considerable eosinophilia (8.6 and 15 per cent., respectively); Malinowski, 3 per cent.

eosinophiles, and Constantin 1 per cent. eosinophiles; but in his case there was a notable polynucleosis.

In two patients (they were brothers) MacCall Anderson found the presence of hæmatoporphyrine in the urine. This substance gave a Burgundy color to the urine. White, Constantin (two cases), Rash, Moller and P. Linser searched for hæmatoporphyrine in the urine and it was found by the last three observers. Linser's case is particularly interesting in this respect. The patient did not present hæmatoporphyrine in the urine excepting during his attacks of hydroa, but when he was subjected to 'X-rays or ultra-violet rays this substance again appeared in the urine.

I would, however, repeat that further researches in this direction are necessary and one cannot, in the present state of the question, draw any conclusions from what few data we have.

The liquid from the bullæ, in the few cases where bacteriological examinations have been made, never gave rise to any growths on culture media.

Hydroa vacciniform almost always occurs a few hours after the patient has been exposed to the sun and more rarely after exposure to a dry, hot wind. The eruption is sometimes preceded or accompanied by some general phenomena, such as malaise, anorexia, nausea, vomiting or chills, but these manifestations are far from being constant.

More commonly the patient complains of sensations of heat, tension or pruritus in the region which will be the site of the eruption. The sites have already been referred to and need, therefore, no further mention. The eruption appears first on the nose and ears in most instances and then spreads to the forehead and neck if the latter has been exposed, and also over the dorsal aspect of the hands. Very infrequently the eruption may become generalized over the back and limbs, and in this case the eruptive elements are invariably few in number. It is also very rare for the eruption to develop on the mucous membranes, but it has been encountered in the mouth, the lips, tongue, conjunctiva, and cornea.

Considered by itself, the eruption begins by more or less prominent red spots (sometimes compared by the patient to flea-bites), or in the form of small, hard subcutaneous nodules upon which vesicles appear almost from the start, while in some cases they appear at once.

At their onset these vesicles have been compared by both Bazin

and Crocker to herpes, which they resemble from their transparency, as well as from their size and distribution, isolated or in clusters on the red spots on the integument. Several elements may coalesce and by this confluence form plaques of variable dimensions and with irregular contours.

The vesicles which are rounded at the onset of the eruption become, according to Bazin, umbilicated about the second day and at the same time lose their transparency. A crust then forms in the centre of these pustules which increases at the periphery and finally falls off, leaving a depressed cicatrix. Bazin also remarks that occasionally the vesicles give rise to a real secretion and the involved areas become covered with thick yellow crusts resembling impetigo.

Let us consider in detail the physiognomy belonging to each eruptive element. At the onset the vesicular projections are formed by small, rounded, globular elevations the size of a millet seed or even a split pea; they are partially transparent or opaline and resist the pressure of the finger. If the element is pricked with a needle a limpid or serous fluid will make its exit, but the epidermic elevation does not collapse, being maintained in shape by the fibrous scaffold formed by the exudate. The vesicles repose either upon a red areola or upon a red, vascularized skin without distinct areolæ.

Occasionally, the element may remain in this state and then dries up, becoming covered by a scab, and at length disappears. But more often it increases in size, growing on the periphery, and attains the volume of a split pea or even larger and its centre then falls in. The element is then umbilicated, the central portion is depressed, dark blue or black, this tint being the result of an interstitial hemorrhage. Around the umbilication the rest of the vesicle forms an elevated opaline collar, itself surrounded by a red areola.

The crust which then begins to form at the centre of the element finally covers it completely by extension of its border. It is deep and adherent, and very dark in color. The crust becomes detached after a variable length of time, usually in about twelve to fifteen days. Its fall exposes the red, depressed, cicatricial dermis, which with time becomes white, leaving a permanent, rounded, white, depressed cicatrix having a vacciniiform aspect.

In one of his cases Bowen noted an enlargement of the cervical and submaxillary lymph-nodes during the eruption. Besides the

eruption properly speaking, the cicatrices resulting from former attacks must be taken into consideration in the symptomatology of hydroa vacciniform.

Besides the lesions actually present in a given case, more or less extensive mutilation of the nose, ears or dorsal aspect of the hands may be noted, which are covered with depressed cicatrices and lines of telangiectasia. The ears particularly may be so severely involved that in some cases they are reduced to their cartilaginous framework and are merely covered by an atrophied skin riddled with cicatrices.

Lastly, certain abnormal localizations are susceptible of giving rise to special symptoms. Thus the extension of the eruption to the buccal or lingual mucosa gives rise to pain, with intense dysphagia sometimes preventing feeding. The corneal manifestations of hydroa vacciniform leave opacities and permanent spots behind.

Hydroa vacciniform does not always offer the typical form just described. In some very few cases the umbilication disappears on account of the intensity of the lesions. In this form the eruption offers an unusual severity in the form of bullæ of varying dimensions, but always considerable, and still more, it does not become strictly localized to the uncovered parts of the integument but manifests a marked tendency to generalization.

White, of Boston, was the first to describe two remarkable examples of this form which some writers hesitated to consider as Bazin's hydroa vacciniform. However, at present this fact is unquestionably admitted and Constantin was able to publish a case of transition between a typical hydroa vacciniform and the form described by White. Constantin proposed to call this clinical type—serious on account of the deep cicatrices and mutilation that is the consequence—*cicatricial bullous hydroa* or *vesiculo-bullous hydroa*, reserving the term hydroa vacciniform for the typical form of the affection.

Moller has also described what he calls *vesiculo-bullous hydroa*, which differs from the typical form of the disease in that it leaves no cicatrices, but this very fact is enough to eliminate it from the hydroa heading and to place it among the recurring summer eruptions.

Each attack of hydroa vacciniform undergoes its evolution in two or three weeks, but since all elements do not develop simultaneously, the entire duration of an attack is somewhat longer. Recurrences are

always possible from the advent of spring to autumn, and they may also occur in sunny days of winter when there is much snow.

Annual recurrences invariably take place; they become less marked at puberty and disappear entirely between the ages of twenty to thirty years.

Hydroa vacciniform has no effect upon the general health, but the number of cicatrices increasing with each renewal of the process finally leads to a more or less considerable disfiguration and in certain forms, especially cicatricial bullous hydroa, the resulting mutilation and deformity of the face or hands is extreme. The prognosis is not good when the cornea is involved because the lesions leave permanent opacities.

The characters of hydroa vacciniform are sufficiently distinct, so that the process need not be mistaken for the majority of the known morbid types. The presence of vacciniform cicatrices and their localization to exposed parts of the cutaneous surface makes the diagnosis certain even when the patient is not suffering from an attack.

The localization of the lesions, their well-known characters will at once eliminate the idea of varicella, a generalized vaccinal eruption or variola. Varioliform or necrotic acne leaves cicatrices similar to those of hydroa, but they are not preceded by umbilicated vesicles and the localization of acne is totally different. The *acnitis* of Barthélmy is located on the hands and face, the lesions occur in successive attacks and leave cicatrices, only the elements of this cutaneous process do not occur in acute attacks as is the case for hydroa. They are at first deep seated, then enlarge, become elevated, the skin reddens over their surface, forms a pustulette which bursts and becomes covered by a little scab, but never forms an umbilicated vesico-pustule.

*Colloid milium* has no umbilication, its development is slow, progressive, chronic. It never occurs in successive acute attacks.

When the vesico-pustules of hydroa are the seat of considerable suppuration, they become covered with yellow impetiginous crusts in which case it might be mistaken for *impetigo contagiosa*, but in these circumstances one will always find some isolated umbilicated lesions characteristic of hydroa. As to the lesions of impetigo, they are always very superficial and never give rise to cicatricial formation.

Pustulous syphilides do not have a predilection for exposed cutane-

ous surfaces and they will probably be accompanied by other evidences of syphilitic infection.

Erythematous lupus involves the nose, cheeks and ears like hydroa vacciniform, but this affection does not develop on surfaces exposed to the solar rays, it does not occur in attacks of vesicles, it does not heal between two consecutive attacks, and the smooth, white cicatrices that it produces in no way resemble those of hydroa vacciniform.

Drug eruptions, principally due to KI, have not the opaline aspect of hydroa, neither are they umbilicated, and in doubtful cases the history will remove all diagnostic hesitation.

In the bullous forms of hydroa a differential diagnosis may have to be made between polymorphous erythema, bullous syphilides, the painful dermatosis of Dühring, pemphigus and hereditary bullous epidermolysis. These various dermatoses are only rarely limited to the usual sites of hydroa vacciniform and none of them leave such marked cicatrices. They do not have annual periodic recurrences during the warm season.

Finally, in congenital bullous epidermolysis, an hereditary affection, the bullæ develop upon the receipt of some kind of traumatism. The action of the solar rays is *nil* in the production of this morbid phenomenon, and what is more, the bullæ are not umbilicated and leave no cicatrices. The differential diagnosis with xeroderma pigmentosum is merely of historic interest and need not detain us.

The treatment of hydroa vacciniform should be above all prophylactic. An endeavor should be made by the use of all possible means to prevent the annual recurrences which are always to be feared at the advent of spring until autumn. There is but one way to attain this and that is to screen the patient from the action of the solar rays.

Many means have been proposed for this end, such as permanently wearing a thick red, yellow or green veil and also protecting the hands with thick gloves. The use of mildly medicated pastes on the face and hands is to be recommended.

Once the vesicular eruption has appeared there is unfortunately little to be done, as no treatment seems to shorten the duration of the attack. Crocker advised to prick the vesicles as soon as they appeared, to empty them and then spray the surface with iodoform dissolved in ether.

Eddowes, from his researches, was led to suspect that the necrosis

of the elements is produced by the pressure of the liquid exudate in epidermis and dermis, and also advises puncture of the vesicles as soon as they appear in order to lower the tension of the exudate and thus prevent cicatricial formation.

When the crusts are loose their fall is hastened by pulverizations and moist clothes, which also have the advantage of quieting the pain, which may be very sharp in bullous hydroa. After the crusts have fallen Audry thinks that it is advantageous to dress the denuded dermis with small compresses moistened with aromatic wine, and this seems to activate cicatrization.

Bazin used to make a great fuss of alkaline treatment and highly recommended the waters of Bourboule, and he may have had good reason for this.

## THE CLINICAL VALUE OF THE WASSERMANN REACTION, BASED UPON A STUDY OF 4500 REACTIONS

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MERCURY possesses an unquestionable effect, both on syphilitic accidents and on Wassermann's reaction. Purkhauer found that the latter became negative after an intensive Hg. treatment in 96 per cent. of the patients so treated; Hoehne, 52 per cent.; Bering and Lesser, 75 per cent.; Blaschko, 87 per cent.; Duhot, 98 per cent., and Mauriac, 75 per cent. On the other hand, in untreated cases of syphilis, Citron obtained 81 per cent. positive reactions and after Hg. treatment this percentage fell to 65 per cent. Neisser caused a drop in positive results in untreated cases from 81 per cent. to 28 per cent.; Bruck, in active secondary syphilis from 87.1 per cent. to 45.1 per cent., in latent secondary syphilis from 50 per cent. to 18.7 per cent., in active tertiary syphilis from 66.6 per cent. to 45.4 per cent., and in latent tertiary lues from 50 per cent. to 16.9 per cent.

However, Gaucher, Paris and Sabaréanu are far from obtaining any such figures as given above and they publish statistics giving 96 per cent. positive results after Hg. treatment. The enormous difference between the above percentages may, however, be explained by the interpretation given to the reaction. In point of fact, in the majority of cases, the reaction is partially influenced and not totally abolished. Therefore, it is classed, according to the viewpoint of the writer, either into positive or negative results. Gaucher, however, does not believe that mercury possesses any important action over the reaction; neither does he believe in the spontaneity of its disappearance any more than from the influence of Hg.

As to Gastou, he attributes a distinct influence to Wassermann's reaction in both the primary and secondary stages of lues, which, in the majority of cases, becomes negative. According to this writer, the results obtained in tertiary syphilis are much less satisfactory.

The influence of salvarsan and neosalvarsan on both syphilitic



accidents and on Wassermann's reaction is still a moot subject. Duhot, Schreiber and Hoppe are of the opinion that its action is so manifest that in the vast majority of cases—almost 100 per cent.—the reaction becomes negative after exhibition of these arsenical products. Other writers maintain that its action varies according to the cases, often of medium intensity and frequently transitory, while the opinion of both Gougerot and Bayet is that the Wassermann is susceptible of frequent returns to the positive. Finally, some writers regard its action as mediocre and almost *nil*. However, Noguchi thinks that these different opinions may be explained from the fact that the reaction is often only partially influenced. The interpretation of the result consequently plays a preponderant rôle, as I have pointed out *à propos* of Hg.

Generally speaking, however, I believe it can be said that all writers recognize that the arsenic products possess a greater influence over the reaction, if not more durable, than mercury. According to Jeanselme and Touraine, the Wassermann will become negative any time between the seventh and fifty-second day following treatment with arsenic. In secondary syphilis these writers have seen it become negative in less than eleven days, in 47 per cent. of their cases. Duhot has noted that the disappearance of the reaction usually takes place between the fourth and eighth week following the treatment, and Joltrain between the sixth day and sixth week and that the result obtained is often of short duration.

Lange found that the reaction became negative between the fourth and fifth week following arsenical treatment and he points out that it usually disappears before the clinical manifestations and rarely remains positive after the specific lesions have subsided. Emery maintains that a distinction should be made between the different periods of syphilis, the change in the reaction being far from the same at the different phases of lues. Thus he has been able to note that in the primary phase it is easy to obtain a durable negative reaction following arsenobenzol treatment. During the secondary period a negative reaction is easy to obtain, but it is often transitory. On the contrary, in the tertiary period a negative reaction is much more difficult to obtain.

I would also point out that quite frequently a Wassermann negative before treatment will become positive after arsenical injections.

This is simply the well-known phenomenon described by Millian under the name of reactivation.

The influence of *mixed treatment* (mercury and arsenic) on the clinical manifestations of syphilis and on Wassermann's reaction is very manifest and it is unquestionable, as I shall show further on, that this is the treatment of choice. The value of this treatment was well known and proven before the war, and Bataille, Payenneville, Loewenberg, not to mention many others, obtained excellent results with it, the relapses in particular appearing to be much less frequent than after the simple arsenical treatment.

The statistics which I shall offer give a percentage of positive results which, in a general way, is much higher than those of other writers, and this is because it has been worked out on an entirely different basis. In general, writers give statistics based on the number of cases that they have treated and whose blood they have examined for two, three, five or more weeks, according to the case. If the Wassermann becomes negative during this lapse of time, the case is recorded as successful; when it is positive it is classed among the failures. From my viewpoint, then, the results thus obtained do not give an exact idea of the question, because it may happen that the Wassermann remains positive for several weeks and only becomes negative much later, at a time when the patient is not even undergoing any treatment at all. On the other hand, it still more frequently happens that a Wassermann becomes negative quite quickly, but that later on the syphilitic manifestations reappear and a positive reaction along with them. In both cases the patient will be attributed to a class to which, in reality, he does not belong.

It is for this reason that I have worked out my statistics, taking as a basis, not the patient himself, but Wassermann's reaction, each patient having had several tests made at various intervals and as regularly as was possible. Consequently, I do not remain within a limited period of a few weeks which follow treatment, but I thus obtain a general view of the evolution of the disease and of the reaction during a number of months or several years. I have thus been able to find that Hg. does not appear to influence the reaction in any marked degree. Generally speaking, the Wassermann remains after treatment what it was before and at the most it is a little weakened. It is practically only in rare cases that the reaction will be found

to disappear, while, on the contrary, it is quite frequent to see it diminish in intensity if recourse be had to the complicated procedures of quantitative analysis. The action of Hg. (see tables A and E) is about the same in the different periods of syphilis, and although I have found a percentage (72.7 per cent.) of positive cases after treatment in tertiary syphilis, below that (87.5 per cent.) of secondary syphilis in the same conditions, I do not conclude from that that Hg. is the cause, because we know that even without former treatment, the Wassermann is much less regularly positive in the tertiary period than during the secondary phase of the infection.

Salvarsan and neosalvarsan have, on the contrary, a very evident action on Wassermann's reaction (see tables B, C and E). This action is particularly remarkable in the primary stage in which the number of positive results following treatment falls to 13.2 per cent. And what is extremely interesting is that in the majority of cases this negative reaction persists for a very long time. In secondary syphilis the action is very marked for a time also. In the very great majority of cases a negative reaction can be obtained without any trouble, but relapses are frequent, and this is what explains the rather high percentage I have found, namely, 53.6 per cent. for 606 and 43 per cent. for neosalvarsan.

In the tertiary period the action of arsenobenzol on the reaction is uncertain and irregular, certain cases are very rebellious to negative transformation; at other times, on the contrary, this transformation takes place with ease, but in a large number of cases I have obtained unstable results; in other words, 47 per cent. and 47.5 per cent. of positive results following treatment.

As to mixed treatment, it has given me by far the best results (see tables D and E). The number of my cases is not large enough to enable me to discuss its effect in each phase of syphilis, but all things taken into consideration, I find that the action of the mixed treatment is still much better than that obtained by salvarsan.

There are figures which speak much more than reasoning, such as those that I have obtained in calculating the *total* percentage of positive Wassermans after different treatments have been given, namely:

After Hg. ....	83.7 per cent.
After 606 and neosalvarsan ....	38.9 per cent.
After mixed treatment ....	29.7 per cent.

This table shows in a most evident way the weak action of Hg., the important action of the arsenical products and the remarkable efficaciousness of mixed treatment.

WASSERMANN'S REACTION IN PATIENTS TREATED WITH Hg.

A.	Syphilis I		Syphilis II		Syphilis III	
Hg. salicylate and biniodide...	1+	0-	87+	13-	25+	8-
Hg. pills.....	1+	0-	14+	0-	7+	2-
Inunctions or gray oil.....	0+	0-	4+	2-	0+	2-
Total.....	2+	0-	105+	15-	32+	12-

WASSERMANN'S REACTION IN PATIENTS TREATED WITH 606

B.	Syphilis I		Syphilis II		Syphilis III	
Without luetic manifestations...	7+	46-	58+	66-	7+	7-
With luetic manifestations.....	...	....	24+	5-	1+	2-
Total.....	7+	46-	82+	71-	8+	9-

WASSERMANN'S REACTION IN PATIENTS TREATED BY NEOSALVARSAN

C.	Syphilis I		Syphilis II		Syphilis III	
Without luetic manifestations...	16+	103-	101+	169-	30+	43-
With luetic manifestations.....	....	....	32+	7-	9+	0-
Total.....	16+	103-	133+	176-	39+	43-

WASSERMANN'S REACTION IN PATIENTS TREATED WITH Hg. AND SALVARSAN

D.	Syphilis I		Syphilis II		Syphilis III	
	3+	6-	7+	18-	1+	2-

PERCENTAGE OF WASSERMANN'S REMAINING POSITIVE AFTER THE VARIOUS TREATMENTS

E.	Syphilis I	Syphilis II	Syphilis III	Total
	Per cent.	Per cent.	Per cent.	Per cent.
Hg.....	....	87.5	72.7	83.7
Salvarsan.....	13.2	53.6	47.0	38.9
Neosalvarsan.....	13.4	43.0	47.5	
Mixed treatment.....	....	....	....	29.7

It is a fact that Wassermann's reaction is always positive in florid secondary syphilis if the patient has undergone no treatment. Therefore, every individual with an untreated syphilis must of neces-

sity have a positive Wassermann at a certain moment in his disease. Is the inverse true? That is to say, will every patient who has not had syphilis present a negative Wassermann? This would be the case unquestionably if the reaction, as was at first thought, was due to the presence of a specific antibody in syphilitic blood, which surely does not exist in individuals free from lues. But it has been demonstrated that Bordet and Gengou's theory on the deviation of the complement, however interesting it may be in itself, cannot be applied to Wassermann's reaction. It is not a case of specific antigens and antibodies, but rather a physico-chemical reaction, at present well understood in its elements, or at all events partially. It is very probably due to the precipitation of lipid substances obtained from the liver of hereditary syphilitics by albumins in the colloid state or globulins contained in excess in the blood of syphilitic subjects.

When these facts became known to syphilographers and were admitted by them, the great enthusiasm raised by the advent of the reaction of fixation gave place to a no lesser scepticism. The majority of the profession then had a tendency to admit that Wassermann's reaction, far from being specific, was met with in the majority of serious affections, capable of producing appreciable changes in the composition of the blood, and not a few writers published cases of positive sero-reaction in various diseases, the two principal ones being scarlatina and leprosy.

It would take up too much space to review or even mention these numerous contributions to medical literature, so that I shall merely refer haphazard to the names of some of these writers *à propos* of each disease. Thus, in scarlatina, Schleisner, Gaucher, Joltrain, Bayet and others obtained positive results, some of them going so far as to pretend that 50 per cent. of cases of scarlatina had the power of deviating the complement. In leprosy, positive results have been recorded by Gaucher and Abrami, Photinos, and Michaelidis, Esmein, Weichselmann, Levaditi, Ledermann, Bayet, Lévy-Bing and Dogny. Spiess and Caan have described cases of cancer with a positive sero-diagnosis. The latter writer obtained a positive Wassermann in thirty-five cancer cases out of a total of eighty-five patients examined. Cutaneous cancers gave him the largest proportion of positive reactions, namely, ten out of fifteen. Gaucher, Paris and Sabaréanu and Hoehne obtained a deviation of the complement in the sleeping sick-

ness. In cases of advanced tuberculosis, Weil and Braun, Ledermann, Lévy-Bing and Dogny, Hoehne and others in fræmbœsia, Eichelberg in diabetes, and Dreyer and Schnitter in saturnism, have obtained the same results. Bar and Daunay have, besides, shown the serum of icterus patients possessed anti-hæmolyzing properties. Some writers have even gone so far as to describe positive reactions in all the infectious diseases: typhoid, malaria, typhus fever, pneumonia and even in anæmia.

I am quite willing to believe that in a certain number of these cases the reactions were carried out with insufficient technical care by men who still lacked experience and practice in the matter, and this is quite comprehensible since the majority of these results date from an epoch when the Wassermann reaction was a recent acquisition. Instead of resorting to the typical Wassermann reaction, the operators often employed some of the derivative procedures in which an antigen of less value was used, such as the extract of normal liver or guinea-pig's heart. And in these researches they, without taking into consideration those cases where, besides the affection incriminated as giving positive results overlooked the possibility of a latent syphilis, often unknown to the patient, and which the writers in their zeal to discover positive Wassermans everywhere, did not try to detect.

This opinion is confirmed by H. Boas' very important paper, which is based upon a study of the reaction in more than two thousand cases, including one thousand and thirty-two control cases divided into ninety-two classes of individuals afflicted by various non-syphilitic processes and which was invariably found to be negative, with the exception of a single case of scarlatina. I will mention only those diseases in which the Wassermann possesses the greatest importance, namely, those in which a positive reaction would seriously involve the value of the method. In sixty-six cases of scarlatina, Boas obtained but one positive result. He, therefore, thinks that in very rare instances and with certain antigens variable according to the patients, scarlatina may give rise to an ephemeral positive reaction. This single positive result does not, according to Boas, decrease the specific value of the Wassermann. In all the other morbid processes Boas found 100 per cent. negative results, namely: in forty-six cases of pulmonary tuberculosis, in forty-six cases of pneumonia, in thirty-two cases of tumors, in thirty-five typhoids and in eleven diabetics. This

writer concludes that in central Europe, given the usual diseases with which the practitioner has ordinarily to deal, a positive Wassermann should be regarded as a sure sign of syphilis. Hoehne is of a like opinion, who obtained only two positive reactions in a total of eleven hundred patients who appeared to be non-syphilitic.

In 1912, Massini published similar results; in all ordinary non-syphilitic processes the Wassermann was invariably negative, but in 1914, Nicolas and Gaté made the statement that they had found 39 per cent. positive reactions in non-syphilitic subjects. There is evidently something abnormal in these results and caused Leredde to say that they could only be explained by some technical error, while Desmoullière pointed out that the results of Wassermann's reaction could not be taken into consideration, because in the same patients in a fortnight's interval and without treatment, Wassermann's reaction was found to change one way or another in 32 per cent. of the cases. This same writer also states that in the experiments carried out by Nicolas and Gaté there was such uncertainty that their results in no way invalidated Wassermann's reaction.

My personal experiments comprise sixty cases, thirteen of the subjects being in good health and forty-seven presenting non-syphilitic affections. Wassermann's reaction was four times positive. One case was a scarlatina in a female forty-five years of age who later on admitted that she had three successive miscarriages and also a non-pruriginous roseoliform eruption. Although this patient had one child in good health, whose birth appears to have been more recent than the roseoliform eruption of the mother, it nevertheless seems to me that her antecedents are sufficiently suspicious to admit the great likelihood of a latent syphilis being present in her case. The same can be said of my second case. This was a male, thirty-four years of age, with serious pulmonary tuberculosis and who admitted that twelve years previously he had had a painless sore which appeared fifteen days after a suspicious coitus and for which he was treated by both an internal and external treatment at Zurich.

The last two positive results concerned two females, respectively fifty and sixty-one years of age, each afflicted with icterus and in both no suspicious anamnestic data could be obtained. I here offer a complete table of my results:

	<i>No. of cases</i>	<i>Negative results</i>	<i>Slightly pos.</i>	<i>Very pos.</i>
Good health .....	13	13	0	0
Scarlatina .....	19	18	1	0
Advanced pulmonary TB. ....	13	12	1	0
Malignant neoplasms .....	9	9	0	0
Diabetes mellitus.....	1	1	0	0
Pneumonia .....	2	2	0	0
Icterus .....	3	1	2	0

Therefore, it will be seen that I never obtained a very positive reaction in any of the above cases. In the four slightly positive cases two can be explained by a possible latent syphilis which is more than likely. The two others were obtained with an icteric serum which, as Bar and Daunay have shown, possesses antihæmolizing properties. All the other results were negative. In cancer and tuberculosis, especially, two affections which syphilis may simulate the most readily, Wassermann's reaction was never positive. Consequently, in non-syphilitic morbid processes it will invariably be negative excepting, perhaps, some affections which are most uncommon in Switzerland and upon which I can form no personal opinion, such as malaria, leprosy, typhus fever, the sleeping sickness, fræmbœsia, etc. However, it would appear that icterus possesses the property of deviating the complement. With this reserve, I conclude that in Switzerland, at least, Wassermann's reaction is practically specific of syphilis.

It has seemed to me interesting, in order to be complete, to follow the evolution and fluctuations of Wassermann's reaction during the progress of syphilis in fifty patients under treatment at the syphilo-graphic clinic and selected absolutely at random. I therefore place myself, for studying the reaction and drawing conclusions, not like the laboratory man, who reasons on the total number of reactions obtained, but as a physician when called upon to make a diagnosis and prognosis, and above all to direct the treatment. A study carried out along these lines may also give numerous complementary data which are not furnished in statistical researches. I have not the pretension, after all the contradictory opinions that have been emitted on this subject, to come to any positive conclusions, but merely wish to publish my observations.

The first few years after the appearance of Wassermann's reaction, all that was required of it was to give diagnostic data, while at the present time it is looked to to settle the prognosis and to direct



the treatment of syphilis. Therefore, it is essential to know if, in reality, the reaction will render these services; in other words, whether the degree of its intensity corresponds with the degree of the patient's infection. To attain good results, many writers have not been content with a qualitative reaction, but have operated by dosing the quantity of the antibody contained in the serum.

Regardless of these improvements in the technic, opinions still vary greatly and the more the question is studied the less clear it appears to become. It is not my intention to mention the names of all the syphilographers who have studied this subject, as the list would be much too long, but for those who desire this information, I would refer them to the very excellent paper published by Lévy-Bing, Dogny and Gerbay in the June, 1914, issue of the *Annales des Maladies Vénériennes*, who give the opinions of numerous writers at the beginning of their paper.

These opinions can be summed up in the following two tendencies: on the one hand, Wassermann's reaction is regarded as the exact index of infection of the organism and as the guide for the prognosis and treatment; on the other, it is looked upon simply as a symptom which may be wanting and which need not be taken into consideration unless it is in accord with the clinical phenomena presented by the patient.

It has often been said that during the evolution of syphilis, Wassermann's reaction undergoes numerous fluctuations, that changes several times from positive to negative, and vice versa, and this without any satisfactory explanation.

Of the fifty cases that I have studied and which, I would repeat, were not selected, twenty-five of them (Cases I to XXV) did not present any variations in the sero-reaction. It is true that I did not resort to the quantitative procedures which are to-day in vogue and I merely contented myself by admitting four degrees, namely: (1) a negative reaction; (2) a slightly positive reaction; (3) a positive reaction, and (4) a very positive reaction. It is therefore certain that minute fluctuations escaped my notice, but I believe that they possess very little value. I endeavored above all to consider the subject in its *ensemble*, because I am of the opinion that mathematical precision does not exist in medicine, no more in the patient's serum

than in laboratory research work. This having been said, I will briefly report the fifty cases studied.\*

CASE I.—Louis P., *æt.* twenty-five years. Diagnosis: Syphilitic chancre (*spirochætæ*) appeared on December 13, 1914.

December 28, 1914: Patient came to clinic with a chancre in the balano-prepuccial furrow. Wassermann —.

December 30, 1914: First injection of neosalvarsan, dose iii.

January 3-22, 1915: Second, third and fourth injections neosalvarsan, doses iv, v and vi.

January 29, 1915: No syphilitic manifestations. Wassermann —.

March 17, 1915: No syphilitic manifestations. Wassermann —.

CASE II.—Arnold Z., *æt.* thirty-six years. Diagnosis: Syphilitic chancre (*spirochætæ*) appeared on April 2, 1914. Came to clinic on April 15, 1914, with several chancres on the balano-prepuccial furrow. Wassermann —.

From April 15th to May 20th was again given six injections of neosalvarsan, doses iv, v, vi, vi, vi, vi.

July 1, 1914: No syphilitic manifestations. Wassermann —.

September 7, 1914: No syphilitic manifestations. Wassermann —.

December 2, 1914: No syphilitic accidents. Wassermann —.

March 19, 1915: No syphilitic accidents. Wassermann —.

CASE III.—Paul G., *æt.* twenty-one years. Diagnosis: Mixed chancres (*spirochætæ*) beginning at the last of November, 1913. Came to clinic on December 17, 1913, with three mixed chancres on glans penis. Wassermann —.

From December 17, 1913, to January 16, 1914, patient was given five injections of neosalvarsan, doses iv, v, vi, vi, vi.

January 23, 1914: No syphilitic accidents. Wassermann —.

May 15, 1914: No accidents. Wassermann —.

July 24, 1914: No accidents. Wassermann —.

CASE IV.—Oscar K., *æt.* thirty-one years. Diagnosis: Syphilitic chancre (*spirochætæ*) began on March 11, 1913. Came to clinic

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\* NOTE.—The different degrees of Wassermann's reaction are represented as follows: Negative reaction (—), slightly positive (sl+), positive (+), very positive (v+).

on March 26, 1913, with a chancre in the balano-prepuccial furrow. Given first injection of neosalvarsan, dose iv.

April 11, 1913: Chancre undergoing cicatrization. Wassermann —. Second injection neosalvarsan, dose v.

April 21, 1913: No syphilitic accidents. Wassermann —.

May 9, 1913: No accidents. Wassermann —.

May 26, 1913: No accidents. Wassermann —.

CASE V.—Charles R., *æt.* twenty-one years. Diagnosis: Syphilitic chancre (*spirochætæ*) appeared in February, 1911. Treated at Berne with four intravenous injections of 606, of 40 and 50 centigrammes. Came to clinic on July 24, 1912, without any syphilitic accident. Wassermann v+. One injection neosalvarsan, dose v.

September 27, 1912: No accidents. Wassermann —.

March 3, 1913: No accidents. Wassermann —.

June 2, 1913: No accidents. Wassermann —.

December 13, 1913: No accidents. Wassermann —.

CASE VI.—Maxime M. Diagnosis: Syphilitic chancre (*spirochætæ*) appeared on December 30, 1912. Came to clinic on January 15, 1913, with a chancre on the frænum. Inguinal adenopathy. Wassermann sl+. Given first injection neosalvarsan, dose v.

February 5, 1913: No syphilitic accidents. Wassermann —. Given second injection neosalvarsan, dose iv.

March 14, 1913: No accidents. Wassermann —.

April 29, 1913: No accidents. Wassermann —.

November 5, 1913: No accidents. Wassermann —.

April 17, 1914: No accidents. Wassermann —.

CASE VII.—Marcel F., *æt.* twenty-nine years. Diagnosis: Tertiary syphilis. Perforation of soft palate. Chancre in August, 1906, treated at Paris with twelve injections of gray oil and Ricord's pills. Came to clinic on June 24, 1913, with destruction of the left portion of the soft palate and two ulcerations on right tonsil. Wassermann sl+. Given first injection of neosalvarsan, dose iv.

July 2 and 11, 1913: Second and third injections of neosalvarsan, doses v and vi.

August 18, 1913: Amelioration of lesions. Wassermann v+.

August 20 and September 3 and 15, 1913, fourth, fifth and sixth injections neosalvarsan, dose v, v, v.

September 29, 1913: Patient in good condition. No accidents. Wassermann v+.

October 6, 1913: Seven injections neosalvarsan, dose v.

December 8, 1913: Wassermann v+. On this day a treatment of fifteen injections of Hg. salicylate was begun.

June 1, 1914: Patient in good health. No accidents. Wassermann v+.

July 22, 1914: Wassermann v+.

November 23, 1914: Began second treatment of fifteen injections Hg. salicylate.

March 5, 1915: No accidents. Good health. Wassermann v+.

CASE VIII.—Giustina F., æt. thirty-four years. Diagnosis: Secondary syphilis without accidents. Roseola and loss of hair in 1913. Treated in Italy with twelve calomel injections. Came to clinic on March 4, 1914. No accidents. Wassermann v+. Began fifteen injections of Hg. biniodide, two centigrammes each.

July 22, 1914: No accidents. Wassermann v+.

July 24, and 31, 1914: Two injections of neosalvarsan, dose iii each.

September 7, 1914, began a series of fifteen injections Hg. biniodide, two centigrammes each.

February 8, 1915. No accidents. Wassermann sl+.

CASE IX.—Henri L., æt. twenty years. Diagnosis: Secondary syphilis without lesions. In the spring of 1912 he developed a curious urticarious eruption and afterwards had intense headache. In August, 1912, there was a sudden falling out of the hair and sore throat. On February 11, 1913, the patient came to the clinic without any syphilitic accident. No trace of any cicatrix could be found on the genital organs or on any other part of the body. Enlarged mastoid lymph-nodes on both sides. Wassermann v+. Given first injection of neosalvarsan, dose iv.

February 26, 1913: Wassermann v+. Second injection neosalvarsan, dose iv.

March 26, 1913: Wassermann v+. Third injection neosalvarsan, dose iv.

April 25, 1913. Wassermann v+. Fourth injection neosalvarsan, dose iv.

May 26, 1913: Wassermann v+.

June 2 and July 16, 1913, fifth and sixth injections, doses v and vi.

August 20, 1913: Patient still without any syphilitic accidents. Wassermann v+. To-day began a series of fifteen injections of Hg. salicylate.

March 2, 1914: Wassermann +.

CASE X.—Antonia C., *set.* thirty-three years. Diagnosis: Secondary syphilis with accidents. Came to clinic with a generalized roseola and headache. Two months pregnant. Wassermann v+. Treatment: Lig. Van Swieten.

June 6, 1913: Generalized roseola, pigmentary syphilides on neck, mucous patches on genitalia. Entered Professor Oltramare's service and there remained until June 17th, during which time she was given two injections (dose iv) neosalvarsan.

November 19, 1913: Eight months' pregnancy. Mucous patches on vulva. Wassermann v+. Third injection neosalvarsan, dose iii.

November 24 and December 1 and 15, 1913, fourth, fifth and sixth injections neosalvarsan, doses iii, iv, iv. Disappearance of lesions.

January 8, 1914: Delivered of a healthy male infant.

December 14, 1914: No accidents. Wassermann v+.

CASE XI.—Vincent V., *set.* twenty-seven years. Diagnosis: Syphilitic chancre. Came to clinic June 2, 1912, with a chancre on prepuce undergoing cicatrization. No spirochætæ could be found. The sore had been dressed with iodoform before the patient was seen; polylymph-nodes adenopathy. Wassermann v+. Given first injection of 606, fifty centigrammes.

July 1, 1912: Complete cicatrization of sores. Wassermann sl+.

September 16, 1912: A small suspicious-looking papule on glans penis. Wassermann v+.

September 17, 1913: No accidents. Wassermann v+.

CASE XII.—Louis B., *set.* nineteen years. Diagnosis: Primary and secondary syphilis; chancre (spirochætæ) and roseola. Came to clinic on March 23, 1913, with chancre in furrow. Commencement of a roseola on body. Wassermann was not done at this time, but would have unquestionably been positive. Given first injection of neosalvarsan, dose iv.

April 4, and 21, 1913: Second and third injection neosalvarsan, dose iv.

May 7, 1913: No accidents. Wassermann —.

June 11, 1913: No accidents. Wassermann —.

July 21, 1913: Patient has complained of persistent headache for the past three weeks. On this day began a series of fifteen injections of Hg. biniodide at the dose of three centigrammes.

October 6, 1913: Headache has disappeared. Wassermann —.

December 10, 1913: For the past fortnight has had mucous patches on glans penis and scrotum. Circinate roseola on thighs. Given fourth injection neosalvarsan, dose iv.

December 17 and 26, 1913, and January 9, 1914, fifth, sixth and seventh injections neosalvarsan, dose vi.

January 14, 1914: No accidents. Wassermann —.

CASE XIII.—Jules G., æt. twenty-six years. Diagnosis: Secondary syphilis. Generalized roseola. Treated in Professor Oltramare's service from December 2 to 28, 1912, for syphilitic roseola. Six intravenous injections of Hg. bichloride, total amount being seventeen centigrammes.

January 3, 1913: Came to clinic. No accidents. Wassermann v+.

January 10, 1913: Return of roseola. Mucous patches on tonsils. Given first injection of neosalvarsan, dose iv.

January 20, 1913: Accidents have disappeared. Wassermann v+.

January 24, 1913: Second injection neosalvarsan, dose iii.

February 24, 1913: Third injection neosalvarsan, dose iii. Wassermann —.

March 26, 1913: Erosion on prepuce the size of an almond, covered by a crust; spirochætæ present. Wassermann —. Fourth injection neosalvarsan, dose iv.

June 30, 1913: Erosive papule on prepuce. Wassermann —.

July 7, 1913: Fifth injection neosalvarsan, dose v.

August 11, 1913: No accidents. Sixth injection neosalvarsan, dose vi. Wassermann —.

January 14, 1914: No accidents. Wassermann —.

CASE XIV.—Joseph B. Diagnosis: Secondary syphilis without lesions. Chancre in 1909. Treated at Paris with ninety Ricord's pills. Married in 1913; wife had miscarriage in the tenth week of pregnancy. Came to clinic on April 8, 1914, for a pruritus ani. Cicatrix of chancre on penis. No other syphilitic lesion. Wassermann v+.

April 15, 1914: Began series of fifteen injections Hg. salicylate.

July 1, 1914: No lesions. Wassermann v+.

July 3 to 24, 1914. Given four injections neosalvarsan, doses v, v, vi, vi.

September 16, 1914: No accidents. Wassermann sl+.

November 4, 1914: Began series of fifteen injections of Hg. salicylate.

January 22, 1915: No accidents. Wassermann —.

April 9, 1915: No accidents. Wassermann —.

CASE XV.—Anna L., æt. eighteen years. Diagnosis: Secondary syphilis with lesions. Chancre at the beginning of July, 1913. Came to clinic on August 29, 1913, with a circular erosion (chancre) on left cheek. Cervical adenitis. Wassermann +.

November 24, 1913: The patient, who had not been seen since August 29th, returned with an erythemato-squamous eruption around the mouth, on the neck and limbs. Mucous patches on vulva.

November 24 to December 15, 1913, was given four injections neosalvarsan, doses iii, iv, v, vi.

December 22, 1913: No lesions. Wassermann v+.

January 19, 1914: Fifth injection neosalvarsan, dose vi.

March 16, 1914: No lesions. Wassermann v+.

March 20 and 27, 1914: Sixth and seventh injections neosalvarsan, dose vi.

June 17, 1914: No lesions. Wassermann —.

October 14, 1914: No lesions. Wassermann —.

CASE XVI.—Alice M., æt. twenty years. Diagnosis: Syphilitic chancre of lip, which appeared on January 10, 1914. Came to clinic on January 30, 1914, with chancre on lower lip, with an indurated base and left-sided submaxillary adenitis. Wassermann sl+. Given first injection neosalvarsan, dose iv.

February 6 and 13, 1914: Second and third injections neosalvarsan, dose v. Chancre had cicatrized.

March 6, 1914: Wassermann sl+.

March 20, 1914: No lesions. Wassermann sl+. Fifth injection neosalvarsan, dose v.

April 3, 1914: No lesions. Wassermann —.

May 1, 1914: No lesions. Wassermann —.

CASE XVII. Emile P., *set.* twenty-two years. Diagnosis: Secondary syphilis with lesions. Chancre? Patient came to clinic on March 19, 1913, with a generalized typical roseola, which had been present for a week. Wassermann v+.

March 19 to April 18, 1913, was given three injections of neosalvarsan, dose iv. The eruption disappeared after the first injection.

May 16, 1913: No lesions. Wassermann sl+.

May 19 and July 2, 1913: Fourth and fifth neosalvarsan, doses iv and v.

September 1, 1913: No lesions. Wassermann —.

December 1, 1913: No lesions. Wassermann —.

CASE XVIII.—Flora S., *set.* twenty-six years. Diagnosis: Syphilitic chancres of urethra. Patient came to clinic on September 16, 1913, with several chancres in the urethral region which had appeared a fortnight previously. Headache. Wassermann sl+. Given first injection neosalvarsan, dose iv.

October 3, 10 and 20, 1913: Second, third and fourth injections of neosalvarsan, dose iv. Disappearance of the lesions.

November 3 and 10, 1913: Fifth and sixth injections neosalvarsan, dose v.

January 2, 1914: No lesions. Wassermann sl+.

February 11, 1914: No lesions. Wassermann —.

May 8, 1914: No lesions. Wassermann —.

June 16, 1914: No lesions. Wassermann —.

CASE XIX.—Ernest S., *set.* twenty-eight years. Diagnosis: Tertiary syphilis. Circinate syphilides. Chancre in 1906. Came to clinic on May 27, 1913, with erythematous-squamous circinate syphilides, in large rings, located on the scrotum and penis. No other lesions. Patient had been treated up to this time with KI only. Wassermann v+. Given first injection of neosalvarsan, dose v.

June 4 and 13, 1913: Second and third injection neosalvarsan, dose v.

June 25, 1913: Disappearance of lesions. Wassermann —.

August 1, 1913: No lesions. Wassermann —.

April 1, 1914: No lesions. Wassermann —.

CASE XX. C. V., *set.* twenty-eight years. Diagnosis: Syphilitic chancre (*spirochætæ*) appeared on January 26, 1911. Came to clinic on February 3, 1911, with chancre in balano-prepuceal sulcus.



Wassermann v+. Given first injection 606, fifty centigrammes intravenously.

March 11, 1911: Chancre cicatrized. Wassermann v+.

April 11, 1911: No lesions. Wassermann sl+.

October 11, 1911: Patient complains of headache. Wassermann v+. Given second intravenous injection of sixty centigrammes 606.

January 8, 1912: Still some headache. Wassermann sl+.

February 12, 1912: No lesions. Wassermann —.

March 25, 1912: No lesions. Wassermann —.

June 19, 1912: No lesions. Wassermann —.

October 3, 1912: No lesions. Wassermann —.

January 30, 1913: No lesions. Wassermann —.

CASE XXI.—Marie C., æt. seventeen years. Diagnosis: Secondary syphilis. Hypertrophic mucous patches on vulva and tongue. Generalized roseola. Treated from May 8 to 23, 1914, in Professor Oltramare's service. Three injections neosalvarsan, doses iii, iv, v. Wassermann v+.

Came to clinic on May 29, 1914, with rapidly retrogressing lesions.

May 29 and June 8 and 15, 1914: Fourth, fifth and sixth injections neosalvarsan, dose v.

June 22, 1914: Disappearance of lesions. Wassermann sl+.

June 24 and July 1, 1914: Seventh and eighth injections neosalvarsan, dose vi.

July 29, 1914: No lesions. Wassermann sl+. Began on this date a series of fifteen injections Hg. benzoate.

October 19, 1914: No lesions. Wassermann sl+.

December 16, 1914: No lesions. Wassermann —.

CASE XXII.—Aloys E., æt. thirty-four years. Diagnosis: Secondary syphilis with lesions. Was treated in Professor Oltramare's service for generalized papular syphilides from May 30 to June 5, 1912. Wassermann v+. First injection (intravenous) of forty-five centigrammes 606.

June 28, 1912: A few papules still present but on the wane. Wassermann v+.

September 4, 1912: No lesions. Given first injection neosalvarsan, dose iv.

September 9, 1912: No lesions. Wassermann v+.

September 18, 1912: Second injection neosalvarsan, dose iii.

June 2, 1913: No treatment since 1912. A return of the roseola for the past two months. Wassermann sl+.

June 3, 11 and 18, and July 2, 1913: Third, fourth, fifth and sixth injections neosalvarsan, doses iv, iv, v, v.

July 9, 1913: No lesions. Wassermann v+. Seventh injection neosalvarsan, dose vi.

November 14, 1913: Wassermann sl+. Began a series of fifteen injections Hg. salicylate.

May 8, 1914: No lesions. Wassermann —.

October 9, 1914: No lesions. Wassermann —.

December 4, 1914: No lesions. Wassermann —.

CASE XXIII.—Hyacinthe B., *æt.* twenty years. Diagnosis: Secondary syphilis with lesions. Was treated in Professor Oltramare's service for a generalized papular syphilide from February 21st to 25th. Wassermann v+. Intravenous injection fifty centigrammes 606. One week later lesions disappeared. Enlarged lymph-nodes in both groins. Pil. Ricord, No. xxx.

July 12, 1912: Enlarged lymph-nodes still exist. Wassermann v+. Second intravenous injection 606, thirty centigrammes.

July 26, 1912: Good general condition. Pil. Ricord, No. xxx.

January 17, 1913: No lesions. Wassermann —.

CASE XXIV.—Arthur S., *æt.* thirty-five years. Diagnosis: Secondary syphilis with lesions. Was treated in November, 1908, for a syphilitic chancre followed by roseola, with KI and six injections of gray oil.

Came to clinic on August 9, 1909, with buccal mucous patches. No eruption on body. Was given a series of fifteen injections of Hg. biniodide, two centigrammes each.

March 10, 1910: Syphilitic angina. Since last seen has taken one hundred and twenty Ricord's pills. Wassermann v+. Given a series of ten injections Hg. salicylate, followed by Pil. Ricord, No. lx.

September 28, 1910: An ulceration on the site of the former chancre. Wassermann v+. Intravenous injection of 606, sixty centigrammes.

June 7, 1911: No lesions. Wassermann v+.

September 6, 1911: No lesions. Wassermann v+.

January 12, 1912: No lesions. Wassermann v+. Began a series of ten injections Hg. bichloride, two centigrammes each.

October 7, 1912: Vertigo. Given first injection neosalvarsan, dose iv.

February 26, 1913: No lesions. Wassermann —. Second injection neosalvarsan, dose iv.

March 28, 1913: No lesions. Wassermann —.

September 5, 1913: No accidents. Wassermann —.

CASE XXV.—Jean V., set. forty-seven years. Diagnosis: Secondary syphilis with lesions. Chancre in June, 1910. Came to clinic on August 1, 1910, with a generalized roseola. Given a series of nine injections Hg. salicylate.

May 31, 1911: Circinate syphilides on lumbar region and left thigh. Wassermann v+. Given first intravenous injection of 606, sixty centigrammes.

July 3, 1911: Disappearance of the lesions. Wassermann v+.

August 2, 1911: No lesions. Wassermann v+.

September 4, 1911: No lesions. Wassermann v+.

October 2, 1911: No lesions. Wassermann v+. Second intravenous injection sixty centigrammes 606.

November 6, 1911: Wassermann v+.

December 4, 1911: No lesions. Wassermann v+. Began series of ten injections Hg. bichloride, three centigrammes each.

February 7, 1912: No lesions. Wassermann v+.

April 10, 1912: No lesions. Wassermann sl+. Began series of ten injections Hg. salicylate.

June 12, 1912: No accidents. Wassermann v+.

July 1, 1912: Wassermann v+.

August 7, 1912: Wassermann v+. Three intravenous injections, fifty centigrammes each, of 606.

September 1, 1912: No lesions. Wassermann v+.

October 18, 1912: First injection neosalvarsan, dose v.

November 20, 1912: Wassermann v+. Second injection of neosalvarsan, dose v.

December 30, 1912: Wassermann v+. Third injection of neosalvarsan.

February 10, 1913: No lesions. Wassermann v+.

April 16, 1913: Fourth injection neosalvarsan, dose iv.

May 18, 1914: Has had no lesions since 1910. Wassermann —.

CASE XXVI.—Jules O., set. thirty-three years. Diagnosis: Ter-

tiary syphilis. Was first treated in 1905, in Professor Oltramare's service, for a papular syphilide. Treatment: fifteen injections Hg. biniodide, two centigrammes each.

April to June, 1913: Second stay in the service for a right-sided specific hemiplegia. Wassermann sl+. Wassermann of cerebrospinal fluid v+. Five injections neosalvarsan, doses ii, iii, iv, iv, iv.

June 6 and 13, 1913: Continued treatment at Polyclinic. Sixth and seventh injections neosalvarsan, doses iv and v.

June 20, 1913: Wassermann —.

August 4, 1913: Good general condition. Wassermann sl+.

August 15 and 25, 1913: Eighth and ninth injections neosalvarsan, dose v.

September 8, 1913: Wassermann sl+.

September 22, 1913: Tenth injection neosalvarsan, dose v.

October 1, 1913: Wassermann —.

November 5, 1913: Good condition. No lesions. Wassermann v+.

November 7 and 14, 1913: Eleventh and twelfth injections neosalvarsan, dose v.

November 21, 1913: Began series of fifteen injections Hg. salicylate.

March 6, 1914: No lesions. Wassermann sl+.

April 14, 1914: Began series of fifteen injections Hg. biniodide, two centigrammes each.

July 24, 1914: Wassermann —.

October 5, 1914: Wassermann sl+. Began series of fifteen injections Hg. benzoate.

January 8, 1915: No lesions. Wassermann —.

CASE XXVII.—Numa Z., æt. twenty-nine years. Diagnosis: Secondary syphilis; alopecia in scalp. Chancre in September, 1910. Came to clinic June 16, 1911, for alopecia; no other lesion. Wassermann v+. Given first intravenous injection, sixty centigrammes 606.

September 27, 1911: Hair growth is general. Wassermann —.

November 10, 1911: Small, suspicious-looking papule on scrotum. Wassermann sl+.

January 10, 1912: No lesions. Wassermann sl+.

February 28, 1912: Typical papule on anal margin. Mucous

patch on left cheek. Wassermann v+. Second intravenous injection fifty centigrammes 606.

September 20, 1912: No lesions. Wassermann v+. First injection neosalvarsan, dose iv.

January 31, 1913: Wassermann v+. Began series of ten injections Hg. salicylate.

January 18, 1915: No lesions for past two years. Wassermann —.

CASE XXVIII.—Arouelo B., æt. nineteen years. Diagnosis: Probable hereditary syphilis.

June and July, 1912, treated in Professor Oltramare's service for a syphilitic gumma with destruction of soft palate. Dental strisæ. Wassermann v+. First intravenous injection thirty-five centigrammes 606. Later, first injection neosalvarsan, dose iv.

December 18, 1912: No lesions. Wassermann v+. Second injection neosalvarsan, dose iv.

February 3, 1913: No lesions. Wassermann —.

March 26, 1913: No lesions. Wasserman sl+.

April 4, 1913: Third injection neosalvarsan, dose iv.

April 18, 1913: Wassermann v+.

May 18, 1914: No lesions. Wassermann —.

CASE XXIX.—Jules B., æt. twenty-nine years. Diagnosis: Syphilitic chancre. Came to clinic October 14, 1908, with a chancre in the balano-prepuccial sulcus. Ten injections of gray oil.

March 17, 1909: Buccal erosions. Began series of twenty injections of gray oil.

December 6, 1910: No lesions. Wassermann v+. Began series of ten injections Hg. salicylate, followed by Pil. Ricord, No. clx.

January 5, 1912: No lesions. Wassermann v+.

February 5, 1912: No lesions. Wassermann sl+.

April 15, 1912: Began Pil. Ricord, No. lx.

September 30, 1912: No lesions. Wassermann —.

February 3, 1913: No lesions. Wassermann sl+.

March 3, 1913: Began Pil. Ricord, No. lx.

April 28, 1913: Wassermann —.

November 10, 1913: Wassermann —.

November 9, 1914: No lesions. Wassermann —.

CASE XXX.—Jean I., æt. twenty-five years. Diagnosis: Sec-

ondary syphilis with lesions. Chancre in October, 1913. Came to clinic on November 18, 1913, with generalized roseola. Cicatrix on meatus of chancre. Inguinal lymph-nodes. Wassermann v+.

November 18 to December 10, 1913: Four injections neosalvarsan, doses iii, iv, v, vi.

January 2, 1914: Began series of fifteen injections Hg. salicylate. Wassermann v+.

March 20, 1914: No lesions. Wassermann —.

May 25, 1914: No lesions. Wassermann sl+.

CASE XXXI.—Augusta M., æt. 29 years. Diagnosis: Active secondary syphilis. Entered service of Prof. Oltramare on Oct. 24, 1911, for papular syphilides of face and trunk. Wassermann v+. First intravenous injection fifty centigrammes 606.

October 30, 1911: Wassermann v+.

November 1, 1911: Disappearance of lesions.

March 17, 1913: No lesion since 1911, no treatment. Wassermann v+. First injection neosalvarsan, dose iii.

April 16, 1913: Wassermann sl+.

April 29 and September 9, 1913: Second and third injections neosalvarsan, dose iii.

March 20, 1914: No lesions. Wassermann sl+.

March 27 and April 3, 1914: Fourth and fifth injections neosalvarsan, doses iv and v.

April 22, 1914: Wassermann —.

May 13, 1914: No lesions. Wassermann sl+.

CASE XXXII.—Charles M., æt. twenty-four years. Diagnosis: Syphilitic chancre. Came to clinic August 12, 1911, with an ulcer in left labial commissure, with indurated edges and covered by a brownish scab. Enlarged submaxillary glands on left side. Wassermann v+. First intravenous injection sixty centigrammes 606.

September 4, 1911: Chancre cicatrized; tonsil large and red.

September 22, 1911: Ulceration of right tonsil. Began Pil. Ricord, No. 1. Five intravenous injections of 606 were given in Paris later on.

September 18, 1912: No lesions. Wassermann —.

December 2, 1912: Wassermann v+. First injection neosalvarsan, dose iv.

March 19, 1913: Mucous patches on tonsils and scrotum. Wassermann v+. Second injection neosalvarsan, dose iv.

April 21 and June 4: Disappearance of lesions. Third and fourth injections neosalvarsan, dose iv.

July 11, 1913: No lesions. Wassermann —.

November 7, 1913: No lesions. Wassermann —.

December 19, 1913: Suspicious erosions on scrotum and glans penis. Wassermann sl+.

December 25, 1913, and January 9, 1914: Fifth and sixth injections neosalvarsan, doses v, vi. The lesions disappeared Jan. 9th.

May 13, 1914: No lesions. Wassermann —.

CASE XXXIII.—Alexandre S., æt. thirty-four years. Diagnosis: Secondary syphilis in activity. Chancre in May, 1912. Came to clinic August 14, 1912, with a generalized roseola of three weeks' duration. Cicatrix of chancre on prepuce. Inguinal adenopathy. Wassermann was not made, but would unquestionably have been positive. First injection neosalvarsan, dose vi.

August 23, 1912: Lesions persist. Second injection neosalvarsan, dose v.

August 26, 1912: Disappearance of lesions. Third injection neosalvarsan, dose iv.

September 27, 1912: Wassermann —.

November 8, 1912: No lesions. Wassermann sl+.

January 1, 1913: Entered Professor Oltramare's service for specific sarcocela. Wassermann v+.

February 7, 1913: Fourth injection neosalvarsan, dose iii.

February 28, 1913: Sarcocela improved. Discharged from service.

March 17, 1913: Came to clinic. Fifth injection neosalvarsan, dose iii.

March 28 and April 11, 1913: Sixth and seventh injection neosalvarsan, dose iii.

April 25, 1913: Wassermann —.

June 4, 1913: No lesions. Wassermann —.

August 22, 1913: Return of circinate roseola on thorax and abdomen, mucous patches in balano-prepuceal sulcus. Wassermann v+. Began series of fifteen injections Hg. salicylate.

September 23, 1913: Erosion on right tonsil, two mucous patches on glans penis.

November 10, 1913: Numerous lesions on glans penis (spirochætæ). Eighth injection neosalvarsan, dose iv.

November 19, 1913: Ninth injection neosalvarsan, dose v.

December 17, 1913: Disappearance of lesions. Tenth injection neosalvarsan, dose vi.

January 2, 1914: Eleventh injection neosalvarsan, dose vi.

January 26, 1914: Wassermann —. Began series of ten injections Hg. salicylate.

May 15, 1914: No lesions. Wassermann sl+.

CASE XXXIV.—Edouard T., æt. nineteen years. Diagnosis: Active primary and secondary syphilis. Began in February, 1913. Entered Professor Oltramare's service on April 9, 1913, with chancre on prepuce and a discrete roseola. Wassermann v+.

April 10 and 17, 1913: First and secondary injections neosalvarsan, dose iv.

April 21, 1913: No lesions. Wassermann v+.

April 25 and July 9, 1913: Third and fourth injections neosalvarsan, doses iv and v.

October 6, 1913: Wassermann —. Fifth injection neosalvarsan, dose v.

November 19, 1913: Three erosions on prepuce, very painful; no spirochætæ; auto-inoculation.

November 22, 1913: Auto-inoculation negative. Wassermann —. Remained in Professor Oltramare's service for ulcer molle (?) until December 8, 1913.

December 22, 1913: Erosions undergoing cicatrization. Wassermann v+.

January 5, 1914: The lesions in the sulcus are not healed; they consist of numerous mucous patches. Intense roseola on trunk. Mastoid lymph-nodes enlarged. Sixth injection neosalvarsan, dose iv.

CASE XXXV.—Marie V., æt. twenty-four years. Diagnosis: Primary, afterwards secondary, syphilis. Began in November, 1910. Chancre on the fourchette and roseola. Treated with fifteen injections Hg. biniodide and fifteen injections Hg. salicylate.

February 10, 1911: Mucous patches in throat. Wassermann v+. First injection (intravenous) sixty centigrammes 606.

March 3, 1911: Disappearance of lesions.

March 15, 1911: Wassermann —.

July 15, 1911: Mucous patches on tonsils (spirochætæ). Some



papules on body. Second intravenous injection forty centigrammes 606.

August 28, 1911.—A hypertrophic patch still persists in the mouth. Began series of fifteen injections Hg. biniodide, two centigrammes each.

October 3, 1911: Icterus for past two days. Wassermann sl+.

December 6, 1911: No lesions. Wassermann —.

January 8, 1912: Mucous patches on left tonsil. Wassermann —. Began series twelve injections Hg. salicylate.

March 11, 1912: Gumma on left thigh Wassermann v+. Began series nineteen injections of Euesol and fifteen injections Hg. benzoate, two centigrammes each.

July 15, 1912: Mucous patches on tongue. Began series of four injections neosalvarsan, dose iii.

January 17, 1913: No lesions. Wassermann —. Fifth injection neosalvarsan, dose iii.

March 3, 1913: No lesions. Wassermann —. Sixth injection neosalvarsan, dose iii.

April 21, 1913. No accidents. Wassermann —.

January 5, 1914: No accidents. Wassermann —.

CASE XXXVI.—Josephine B., æt. twenty-four years. Diagnosis: Active secondary syphilis. Came to clinic on February 27, 1914, with generalized papules syphilides dating back for one month. Mucous patches on tonsils and vulva. Wassermann v+.

February 27 to March 27, 1914: Five injections neosalvarsan, doses ii, iii, iv, v, v. Disappearance of lesions.

April 3, 1914: Wassermann —.

September 30, 1914: No lesions. Wassermann —.

January 8, 1915: Return of mucous patches on vulva. Wassermann v+.

January 8 to 22, 1915: Sixth, seventh and eight injections neosalvarsan, doses iii, iv, v.

February 12, 1915: Disappearance of lesions. Wassermann —.

CASE XXXVII.—Juliette B., æt. forty years. Diagnosis: Active secondary syphilis. Chancre in March, 1910. Generalized roseola in April, 1910 (Wassermann not done). Pil. Ricord, No. lx. Eleven injections Hg. salicylate and fifteen injections Hg. biniodide, two centigrammes each. KI.

November 9, 1910: No lesions. Wassermann v+. Began series of ten injections Hg. salicylate.

December 12, 1910: No lesions. Wassermann sl+. Began KI, two grammes daily, and Pil. Ricord, No. cx. This treatment ended on August 23, 1911.

October 20, 1911: Some headache. Wassermann v+. First intravenous injection sixty centigrammes 606.

November 29, 1911: Headache persists. Wassermann sl+.

February 12, 1912: No lesions. Began treatment with Pil. Ricord, No. clxx.

February 19, 1913: Headache. KI, two grammes daily.

April 16, 1913: Headache. Wassermann —.

December 10, 1913: No lesions. Wassermann v+.

January 7 to 28, 1914: Three injections neosalvarsan, doses iv, v and v.

March 4, 1914: Wassermann sl+. Pil. Ricord, No. l.

February 8, 1915: No lesions. Wassermann sl+.

CASE XXXVIII.—Hilda S., æt. twenty-eight years. Diagnosis: Active secondary syphilis. Treated in Professor Oltramare's service from October 28 to November 21, 1908, for mucous patches on vulva and generalized roseola. Twenty inunctions Hg. ointment.

December 9, 1908: Returned to clinic with mucous patches on anus. Pil. Ricord, No. xxx.

February 3, 1911: No lesions. Wassermann —. First intravenous injection sixty centigrammes 606.

February 18, 1911: No lesions. Wassermann v+.

November 4, 1912: No lesions. Wassermann —.

December 11, 1912: No lesions. Wassermann —.

February 19, 1913: No lesions. Headache. Wassermann sl+. Pil. Ricord, No. xxx.

CASE XXXIX.—Charles G., æt. twenty years. Diagnosis: Syphilitic chancre (spirochætæ). Began June 15, 1914. Came to clinic on June 22, 1914, with a chancre in the balano-prepuceal furrow with indurated base.

June 22 to July 22, 1914: Five injections neosalvarsan, doses iv, v, v, vi, vi. Cicatrization of chancre after fourth injection.

July 29, 1914: Wassermann sl+.

August 31, 1914: Began series of fifteen injections Hg. salicylate.

November 9, 1914: No lesions. Wassermann —.

January 25, 1915: No lesions. Wassermann —.

CASE XL.—Jean V., *set.* thirty-nine years. Diagnosis: Secondary syphilis with lesions. Chancre in January, 1911. Came to clinic on July 10, 1911, with a generalized roseola. Wassermann v+. First intravenous injection sixty centigrammes 606.

August 30, 1911: No lesions. Wassermann —.

October 4, 1911: No lesions. Enlarged mastoid lymph-nodes. Wassermann v+.

November 6, 1911: No lesions. Wassermann v+.

January 3, 1912: No lesions. Wassermann v+.

February 5, 1912: No lesions. Wassermann v+.

March 19, 1913: Ulcero-squamous lesions appeared on glans penis and scrotum. Wassermann v+. First injection neosalvarsan, dose iv.

April 3, 1913: Disappearance of lesions. Second injection neosalvarsan, dose iv.

April 30, 1913: No lesions. Wassermann sl+.

CASE XLI.—Armand B., *set.* twenty-six years. Diagnosis: Primary syphilis (not diagnosticated) and active secondary syphilis.

December 30, 1912, to February 20, 1913: Treated in Professor Oltramare's service for a soft phagedemic chancre, January 20th and 30th, Wassermann v+.

March 19, 1913: Complete cicatrization of chancre. Came to clinic with a discrete roseola which appeared six days previously. March 19th and 26th, two injections neosalvarsan, dose iv.

April 4, 1913: No lesions. Wassermann —.

April 23, 1913: Third injection neosalvarsan, dose iv.

May 30, 1913: No lesions. Wassermann —. Fourth injection neosalvarsan, dose iv.

July 2, 1913: No lesions. Wassermann —.

August 19, 1913: No lesions. Wassermann sl+.

CASE XLII.—Evaldo G., *set.* eighteen years. Diagnosis: Syphilitic chancre and mucous patches.

From August 27, 1906, to August 16, 1909, patient had received as treatment thirty injections of Hg. salicylate, KI, thirty intravenous injections Hg. bichloride, Pil. Ricord, No. lx, and forty injections Hg. salicylate.

August 16, 1919: Mucous patches in mouth. Began series of thirty-five injections Hg. salicylate.

December 7, 1909: Wassermann v+.

August 7, 1911: No lesions. Wassermann —.

November 10, 1911: No lesions. Wassermann —.

March 10, 1913: No lesions. Wassermann —.

June 4, 1913: No lesions. Wassermann sl+.

September 1, 1913: No lesions. Wassermann —.

December 29, 1913: No lesions. Wassermann —.

CASE XLIII.—Léon G., *æt.* twenty-four years. Diagnosis: Syphilitic chancre (*spirochætæ*). Came to clinic on February 21, 1912, with a chancre in the balano-prepuccial fold. Enlarged inguinal lymph-nodes on right. Wassermann sl+. First intravenous injection fifty centigrammes 606.

March 18, 1912. Chancre healed. Wassermann v+.

May 13, 1912: No accident. Wassermann —.

June 14, 1912: No accident. Wassermann sl+.

Wassermanns made on July 22, August 30, October 14, December 18, 1912, and April 13, 1913, and January 13, 1914, were all negative. No lesions.

CASE XLIV.—Joseph N., *æt.* twenty-one years. Diagnosis: Syphilitic chancre, mucous patches on tonsils.

From August, 1906, to November, 1910, followed a continued treatment. Was given one hundred and eighty injections Hg. salicylate and Pil. Ricord, lx in all. The mucous patches disappeared in January, 1907.

November 21, 1910: No lesions. Wassermann v+.

March 20, 1911: Began series of ten injections Hg. salicylate.

October 23, 1911: No lesions. Wassermann sl+.

November 27, 1911: No lesions. Wassermann v+. Began series of ten injections Hg. salicylate.

January 5, 1912: No lesions. Wassermann v+.

September 2, 1912: No lesions. Wassermann v+.

March 31, 1913: No lesions. Wassermann v+.

April 4 and 16, 1913: Two injections neosalvarsan, doses iii and iv.

April 28, 1913: Wassermann —.

June 11, 1913: No lesions. Wassermann —.

September 19, 1913: No lesions. Wassermann sl+.

December 29, 1913: No lesions. Wassermann —.

May 1, 1914: No lesions. Wassermann —.

CASE XLV.—Marie C., *æt.* twenty-five years. Diagnosis: Active secondary syphilis. Treated in Professor Oltramare's service from February to April, 1911, for a generalized papular syphilide. Wassermann v+. First injection sixty centigrammes 606 intravenously. Left the service with mucous patches still present.

March, 1912: Second stay in service for return of roseola. Wassermann v+. First injection neosalvarsan, dose iv. Discharged without lesions.

April 4, 1913: No lesions. Wassermann +. Second injection neosalvarsan, dose iv.

June 4, 1913: Third injection neosalvarsan, dose iv.

July 4, 1913: Wassermann sl+.

August 22 to September 24, 1913: Fourth, fifth and sixth injections neosalvarsan, doses iv, v, v.

November 7, 1913: No lesions. Wassermann —.

February 6, 1914: No lesions. Wassermann —.

June 8, 1914: No lesions. Wassermann —.

December 18, 1914: No lesions. Wassermann sl+.

February 12, 1915: No lesions. Wassermann —.

CASE XLVI.—Joseph T., *æt.* thirty years. Diagnosis: Syphilitic chancre. Papular syphilide on trunk. Mucous patches on tonsils. February 28, 1905, to January, 1910, continuous treatment. Patient was given in all: one hundred and twenty injections Hg. salicylate, forty (two centigrammes) injections Hg. biniodide, ten injections of gray oil, Pil. Ricord, No. cxx, and KI. No lesion since November, 1906.

January 10, 1910: No lesions. Wassermann v+.

March to the end of August, 1910: Pil. Ricord, No. xl, and ten injections Hg. salicylate.

October 10, 1910: Specific (?) rhinitis. Wassermann v+.

November 18, 1910: Wassermann v+. Two grammes KI daily.

February 20, 1911: Began series of ten injections Hg. salicylate.

April 3, 1911: No lesions. Wassermann v+.

April 21, 1911: Ozena. First intravenous injection sixty centigrammes 606.

August 21, 1911: No odor from nose. Wassermann —.

September 25, 1911: Nasal odor increased. Wassermann +.

September 27, 1911: Nocturnal headache. Second intravenous injection sixty centigrammes 606.

October 23, 1911: No lesion. Wassermann —.

November 29, 1911: No lesion. Wassermann —.

December 1, 1911: Patient complains of bad taste in mouth. KI, two grammes daily.

February 28, 1912: Perforation of nasal septum. Wassermann alt. KI.

August 5, 1912: Cicatrization of nasal lesion. Wassermann —.

First injection neosalvarsan, dose iv.

September 6, 1912: Ozena persists. Second injection neosalvarsan, dose vi.

February 5, 1913: Slight ozena. Wassermann —. Third injection neosalvarsan, dose iv.

March 12, 1913: No lesions. Wassermann —.

May 12, 1913: Idem. Idem.

June 25, 1914: Idem. Idem. KI, two grammes daily.

September 29, December 8, 1914, and July 24, 1915: No lesions. Wassermann —.

CASE XLVII.—Paul B., set. twenty-three years. Diagnosis: Syphilitic chancre (*spirochætæ*). Came to clinic on April 10, 1911, with chancre on prepuce. Inguinal adenopathy. Wassermann v+. First intravenous injection sixty centigrammes 606.

May 12, June 25 and August 11, 1911: No accidents. Wassermann —.

October 21, 1911: For two weeks roseola on body and limbs. Wassermann v+. First injection neosalvarsan, dose iv.

November 13, 1911: Roseola disappeared. Wassermann v+. Second injection neosalvarsan, dose iv.

February 24, 1913: Suspicious erosion on tonsil. Wassermann —. Third injection neosalvarsan, dose iv.

April 4, 1913: No lesions. Wassermann —.

July 4, 1913: No lesions. Wassermann v+.

August 29, 1913: Return of circinate roseola on trunk. Began series of fifteen injections Hg. salicylate.

December, 1913: Fifteen injections Hg. salicylate.

January, 1914: No accidents. Began series of fifteen injections Hg. salicylate.

March 11, 1914. Wassermann v+.

April 15, 1914: Return of circinate roseola. Fourth injection neosalvarsan, dose iv.

April 24 to May 20, 1914: Fifth, sixth, seventh and eighth injections neosalvarsan, doses v, vi, vi, v.

July 22, 1914: No lesions. Wassermann —.

October 23, 1914: Idem. Idem.

March 24, 1915: No lesions. Wassermann sl+.

CASE XLVIII.—Michel B., *set.* thirty-six years. Diagnosis: Secondary syphilis, mucous accidents. Came to clinic on August 21, 1908, with mucous patches on tonsil. From this date to October 31, 1910, patient was given thirty injections of gray oil, Pil. Ricord, No. xxx, and KI almost continuously.

October 31, 1910: No lesions. Wassermann v+. Five injections of gray oil.

December 2, 1910: Wassermann sl+.

February 6, 1911: No lesions. Wassermann —.

June 12, 1911: No lesions. Wassermann —. Five injections of gray oil.

December 20, 1911: No lesions. Wassermann v+.

February 5, 1912: No lesions. Wassermann v+. Began series of fifteen Hg. salicylate injections.

September 30, 1912: No lesions. Wassermann —. Began series of ten Hg. salicylate injections.

January 3, 1913: No lesions. Wassermann v+.

February 10, 1913: Began series of ten Hg. salicylate injections.

June 9, 1913: No lesions. Wassermann —.

September 12, 1913: Suspicious erosion on buccal mucosa. Wassermann sl+.

September 13, 1913, to March 14, 1914, was given eight injections neosalvarsan, doses iv, iv, v, v, iv, v, v, v, and ten injections Hg. salicylate. Lesion disappeared on November 14, 1913.

March 13, June 17 and November 18, 1914: Wassermann —. No lesions.

CASE XLIX.—Marcel P., *set.* twenty-six years. Diagnosis: Secondary syphilis without lesions. Chancre in 1910. Ulcerations on

scrotum in same year. Patient's wife had a child in 1911, presenting distinct lesions of hereditary syphilis. Came to clinic on April 2, 1913, without lesions. Wassermann —. Patient's wife gave a very positive Wassermann reaction.

April 7, 1913: Wassermann —. First injection neosalvarsan, dose ii.

May 14, 1913: Second injection neosalvarsan, dose iii.

May 21, 1913: Wassermann —.

June 2, 1913: Third injection neosalvarsan, dose iv.

June 27, 1913: No lesions. Wassermann sl+. Fourth injection neosalvarsan, dose v.

July 14, 1913. Wassermann —.

August 18, 1913: Wassermann sl+.

September 5, 1913: Headaches. Fifth injection neosalvarsan, dose v.

September 19, 1913: No lesions. Wassermann sl+.

March 10, 1914: No lesions. Wassermann —.

CASE L.—Alfred G., *et.* thirty-five years. Diagnosis: Active secondary syphilis. Chancre in 1910. Came to clinic on May 30, 1910, with a papular syphilide of scalp. Inguinal adenopathy on right side. Wassermann +. Began series of fifteen Hg. salicylate injections.

August 26, 1910: Erosions on tonsils. KI.

October, 1910: Series of fifteen Hg. salicylate.

November 25, 1910: No lesion. Wassermann +.

January 19, 1911: Began series of thirty Hg. salicylate injections.

September 18, 1911: Wassermann v+. Began series of twelve injections Hg. salicylate.

January 8, 1912: No lesions. Wassermann v+. Began series of fifteen Hg. salicylate injections.

May 6, 1912: No lesions. Wassermann —.

June 10, 1912: No lesions. Wassermann v+. Began series of fourteen injections Hg. salicylate.

September 27, 1912: Wassermann —. Began series of fifteen Hg. salicylate injections.

January 13, 1913: Wassermann v+. First injection neosalvarsan, dose iii.

January 27, 1913: Second injection neosalvarsan, dose iii.

March 10 and October 24, 1913: No lesions. Wassermann —.



Of my fifty cases, forty-one (Cases I to XLI) presented a logical evolution of their Wassermann. In all these patients there is not a reaction which cannot be accounted for. Cases XLII to XLV might also be included among them, because, in point of fact, in these four cases only once could the Wassermann reaction not be clearly explained. This was a positive reaction occurring in the midst of a series of negative results after the treatment had been completed. In this case it must be admitted—a thing that is very possible—that there was a sort of congestive attack, similar to those encountered in tuberculosis. By this I mean a momentary awakening of the syphilis and leaving no trace afterwards.

Among these forty-one patients presenting logical Wassermans, many differences are nevertheless noted. The first six (Cases I–VI) are instances of aborted primary syphilis. In the first four the Wassermann was made so early in the process that it was still negative, and the patients received an energetic treatment with neosalvarsan. In these six cases the reaction was invariably negative. On the contrary, in the following five (Cases VII–XI) the syphilis was stubborn in character and all treatments resorted to were powerless to bring the Wassermann round to negative, although they acted favorably on the specific accidents themselves. These are instances of what Milian calls *irreducible* syphilis. Cases XI and XXV are instances where the intensity of the reaction progressively decreased, but with a greater or lesser rapidity. I will not certify that decrease was always absolutely progressive, because I did not possess exact quantitative data, but recurrences, if they did take place, may be regarded as practically negligible.

Patients XXXVI–XLI presented notable and very often numerous variations in their Wassermans, but these variations could always be very easily accounted for on account of their close relationship to the treatment. In most of the cases they were patients who reached a negative reaction and who, left to themselves without treatment, relapsed and returned to the Polyclinic with a positive Wassermann, accompanied or not by new lesions. At other times the interpretation of these variations should be looked for in Milian's phenomenon of reactivation (Cases XXXVIII, XXXVII, XXXIX and XLIII).

Finally, the last nine cases (Cases XLII–L) presented variations in their reactions which cannot be explained. These variations were

always in one of the reactions (+ or -) and after treatment, in cases XLII to XLVI, and in several reactions during treatment in the last five patients (Cases XLV-L).

In a general way, antiluetic treatment is more distinctly effective and acts early on the specific lesions than on the Wassermann reaction. Salvarsan itself, no matter what has been said about this preparation, attacks the lesion and cicatrizes it before it causes the sero-reaction to disappear.

The cases here given offer numerous examples of these facts (Cases VII to XI, XIII to XVIII, XXIV, XXV, XXVIII, XXX, XLIII, etc.). Cases in which Wassermann's reaction became negative before the accidents had subsided (XXXVIII-XLVI) or even remained negative during a new syphilitic outburst, are the exception. These data obtained by clinical observation demonstrate the very little credit which should be given to the theory that Wassermann's reaction after treatment should be negative on account of the presence of arsenic salts or Hg. in the patient's blood. Experiments *in vitro* have been resorted to by many writers in relation to this subject, and the results obtained have been very far from conclusive.

Dobri and Brück, the first to have made experiments in this direction, showed that mercurial, arsenical or iodide solutions have no action on the sero-reaction. Joltrain, experimenting with colloidal Hg., "never observed any changes in the results obtained with Wassermann's reaction. It is quite an impossibility to test, from this point of view, all the mercurial salts which, for the most of them, really possess a slight hæmolytic power, and particularly is this the case with the cyanide." Then, turning his attention to colloidal arsenic (sulphide of arsenic), Joltrain found that "the reaction was not changed." "On the contrary, Epstein and Pibram prevented the reaction from taking place, say Gougerot and Parent, by adding five centigrammes  $\text{HgCl}_2$  to a fixing serum: the same dose of  $\text{HgCl}_2$  with some serum of any kind, is not hæmolyzing." Other writers then took up the subject and came to the same conclusions, Satta, Donati and Brück in particular. Experiments with arsenic salts have not been any more decisive.

My cases demonstrate, I think, that this theory, which maintains that the Wassermann is modified by the presence of medicamentous substances in the patient's blood, should be discarded until more exact

data have been obtained. From the practical standpoint, Hg. and arsenic modify the reaction by their curative virtues; that is to say, by the more or less complete destruction of the spirochæta, and the very numerous instances where the treatment brings about a negative reaction which later becomes positive again, in no way invalidate this theory. They simply prove that the patient was not cured and that he is a victim of a relapse (Cases XXVI to LXI).

I have tried to show that Wassermann's reaction usually remains positive after cicatrization of the specific lesions, but it may also announce the occurrence of fresh syphilitic outbursts, and although such cases are not frequent, they are, nevertheless, encountered from time to time. Thus it was in Case XLVI. It will be seen that on the date of July 4, 1913, the patient presented no lesion whatsoever, but the reaction was very positive and announced the return of a circinate roseola, which developed on August 29th of the same year. These data, which appear to belong to secondary syphilis are, however, met with in the tertiary phase of the infection. In Case XXXIII, it will be seen that a positive reaction appeared in the month of November in a patient in good health and this reaction was the forerunner of a tertiary lesion (specific sarcocele) which only developed on January 1, 1913, but which certainly existed much before that date, although clinically not evident.

An examination of these fifty cases likewise corroborates conclusions that I had come to from a statistical study of the results in relation to the influence of the various treatments on Wassermann's reaction. First, mercury has little influence on this reaction (Cases VII, VIII, XIV, XXIV, XXXV, XXXVII, XLII, XLIV and XLVI); nevertheless, in certain cases following a prolonged Hg. medication, more or less durable negative reactions may be obtained (Cases XXIX, XXXVIII, XLVIII). Secondly, salvarsan and neo-salvarsan possess a manifest action on Wassermann's reaction (Cases XII, XV, XVI, XVII, XVIII, XX, XXVIII, XLI, XLIII, XLV and XLVII), usually durable in primary syphilis (Cases I to VI), but often temporary in other phases of syphilis, above all in the secondary period (Cases XXVII, XXXIII, XXXVI, XL and XLVII). Thirdly, the mixed treatment is the most efficacious and is often successful where others fail (Cases IX, XXI, XXII, XXIV, XXVII, XXX, XLIV, XLVI and L). This is the opinion of vari-

ous writers, among others, Lévy-Bing and Durcœur, who believe that 606 cures the lesions but does not cure the infection. In order to battle against lues with the maximum efficaciousness, Hg. and the arsenical products must be combined. Nevertheless, in certain cases success is not forthcoming, in spite of varied and intensive treatments in obtaining a negative reaction (Cases VII to XI), and simply diminish the intensity of the reaction. At other times, the reaction which remained frankly positive during treatment, becomes negative of itself some weeks later at a time when all therapeutic interference has ceased. Such was the case particularly in patients representing Cases XXI and XXV.

The variations of Wassermann's reaction during treatment of syphilis are at present admitted by all syphilographers, and it is only in exceptional cases the reaction is observed to become progressively negative. I have already said what should be thought of these oscillations when they are weak and only to be detected by the new quantitative procedures, so that I shall not return to their consideration. As to oscillations of great amplitude, I have seen them in few cases (Cases XLII to L); they cannot be explained, and are data of observation only. Lévy-Bing, Dogny and Gerbay have published beautiful examples, and already in 1912, Leredde and Rubinstein, in a paper read at the *Société Française de Dermatologie et Syphilographie*, insisted on this irregular evolution of Wassermann's reaction. They point out that, in general, the reaction goes through three successive phases. There is the first period during which the reaction remains very positive, a second period of irregularities and a third of negative Wassermann. Be it noted that the latter period is frequently not durable, relapses being frequent. I have, however, noted that the great oscillations are especially encountered in subjects who have undergone no treatment during the primary and secondary stages of the infection (Case XLIX) or who have only been treated with Hg., even intensively (Cases XLVI, XLVIII and L). They are infinitely more rare, at least in that degree of amplitude extending sometimes from a distinct negative reaction to a very positive one, in patients who, from the very start, have been treated with salvarsan. Such instances are, however, met with in the latter cases (Case XLVII).

The *diagnostic value* of Wassermann's reaction unquestionably de-

depends upon its specificity and writers either accord it a very great importance, or, on the contrary, surround it with the utmost scepticism, according to the results they have obtained with Wassermann's reaction during syphilis or in other morbid processes. Boas, who met with a positive reaction in non-syphilitic affections only once out of a total of 1032 cases examined, accords it great credit. Nicolas and Gaté, on the contrary, who obtained 39 per cent. positive results in patients without syphilis, greatly doubt the value of the data that the reaction may furnish. I have already stated what I believe should be thought of results so differently regarded by various writers.

Writing on the subject, Gastou and Lebert state that Wassermann's reaction possesses considerable diagnostic value, and I am likewise of this opinion. This opinion is based upon the results arrived at by the majority of observers, my own experience and the conclusions I have formulated in other writings. I maintain the *practical specificity* of Wassermann's reaction. In point of fact, this reaction is never positive in tuberculosis or cancer, any more than it is in the general diseases common to Switzerland; it is, however, possible and even probable that it is often positive in certain diseases which are rare in Switzerland, such as leprosy, malaria, the sleeping sickness, etc., and icterus (Cases IV and XXXV). These causes of mistake once eliminated, Wassermann reaction when positive can be looked upon as a certain sign of syphilis.

From the practical viewpoint, the diagnostic value of the sero-reaction is different in the various phases of syphilis. In the primary phase it is practically *nil*. The reaction, as I have pointed out, becomes, on an average, positive only on the fifteenth to the twentieth day following the appearance of the primary sore and before then the search for the sero-reaction has no value whatsoever. The clinical signs and above all search for the spirochæte are otherwise important.

During the secondary period in activity, the Wassermann gives 100 per cent. positive results if the patient has undergone no treatment; therefore, it possesses a high diagnostic value when the physician is in doubt about a roseoliform eruption of uncertain pathogenesis. But, generally speaking, the anamnesis and clinical findings are sufficient for the diagnosis and the Wassermann often superfluous.

It is not the same in the tertiary and quaternary periods, as well as in hereditary syphilis, where the manifestations of the disease are

so varied that the diagnosis is frequently uncertain. A positive Wassermann will often be the only means to demonstrate the specific nature of a given lesion. Unfortunately, at these stages of syphilis, the Wassermann is not constant and will be found negative in about 20 per cent. of patients who have not been treated. From this fact, and this is recognized by all syphilographers, it results that a negative reaction in a case of tertiary, quaternary or hereditary syphilis is devoid of any value.

To sum up, it may be said that if a positive Wassermann is in practice synonymous of syphilis, a negative Wassermann does not indicate that syphilis should be eliminated from the diagnosis.

The value of Wassermann's sero-reaction in the *prognosis* of syphilis is a very moot subject and greatly discussed. For some it is important, *nil* or very trifling for the majority of observers. Blumenthal accords no prognostic significance to the reaction, at least during the first few years of syphilis. Gougerot also believes that the Wassermann has no prognostic value, maintaining that it is merely a symptom which should be interpreted along with the other clinical manifestations. Paris and Sabareanu come to about the same conclusions. These observers have frequently observed a generalization of the treponema occurring very shortly after a negative reaction had been obtained. They believe that a negative Wassermann does not even indicate that the case is one of attenuated infection. According to Gaston and Girauld, "a positive sero-reaction indicates that the syphilis may be on the way to activity, but it in no manner indicates—given the modern procedures—the gravity or extent of the affection."

Nevertheless, several German observers are of the opinion that a series of negative reactions means a good prognosis and indicates that a recurrence is not to be feared. Leredde and Rubinstein also believe that the sero-reaction may serve as an index in prognosis. They have noted that if at the time of the secondary period the reaction is weakly positive, the syphilis will be rather mild and will yield easily to treatment.

To my mind, a single Wassermann reaction has no prognostic value whatsoever, but this is not the case if a series of reactions give the same result. In the latter circumstance, two very different aspects

are to be distinguished, namely, the immediate prognostic value and the future prognostic value. In point of fact, the sero-reaction may give useful data on the immediate prognosis, and I believe that even after an energetic treatment, when the Wassermann remains positive for some time (Cases VII and XI) one should fear an early recurrence and that the continued positive results is a sign of a stubborn syphilis, difficult to control. On the contrary, a Wassermann which quickly becomes negative at the same time that the clinical manifestations of the infection disappear, which remains negative and especially does not offer the phenomenon of biological reactivation, eliminates the probability of a near recurrence (Cases V and VI).

On the other hand, the Wassermann reaction has no value in the future prognosis of the infection. Cases of late recurrence after a series of negative Wassermans are far too numerous, it seems to me, to cause any doubt to subsist. And this brings me to speak of the so-called cases of cure of syphilis by abortive treatment. I believe that if the physician, from the purely scientific viewpoint, can call such cases cures, from the social viewpoint, in the present state of our knowledge, it is his duty never to affirm it. The subject is still too new for an evident demonstration of cure to be made, and so far as I am concerned I am persuaded that a quantity of so-called cured syphilitics will sooner or later suffer from relapses in the form of tertiary manifestations or parasyphilis.

The value of Wassermann's reaction as a *guide to treatment* is a problem which, although it is being carefully studied, is yet far from being solved. Some observers, Germans for the most part, rely on the sero-reaction for treatment of the case, as if it were always the exact expression of the degree of the syphilitic infection; others place less reliance on the results of the Wassermann, regarding the reaction as an adjuvant symptom, and conduct the treatment according to the impression they have of the case at the time, or base their conduct on certain rules they have adopted. Finally, there are others who make use of the luetic manifestations in order to prove the efficaciousness of some line of treatment to them personal, and this tendency is particularly evident in regard to the abortive treatment of syphilis.

It is certain that in treating syphilis at the very beginning of the chancrous phase and if possible while the Wassermann is yet nega-

tive, either by injections of gray oil as recommended by Dubot, or by a local treatment by injections of bectine and Hg. as is Hallopeau's practice, or by salvarsan, the result will be that in the majority of cases the reaction will remain negative for many months or even several years, from whence this conclusion of not a few observers that the syphilis is permanently cured. Such is the opinion of Guiard, Leredde, Milian, Jeanselme and Duhot, not to mention many others, who, nevertheless, before affirming that a cure has been attained, submit the patient to the reactivation test of Emery and Netter, who maintain that the infection can be eliminated if treatment is begun in the primary phase of the disease and that there are 70 per cent. to 80 per cent. chances of cure in the secondary period. And the opinion of these observers is still more confirmed by the numerous instances of syphilitic reinjection published in all countries. Let it be noted, although I shall not insist upon it as it is not within the province of this paper, that the majority of these reinfections are subject to suspicion, that frequently the supposed first infection may very well have not been syphilitic or that the so-called second infection may simply be a chancriform eruption.

In this respect all observers are not ready to admit the fact of a permanent cure of syphilis so quickly and several have recorded cases which tend to prove that often this cure is only apparent. Gaucher and Joltrain believe that the abortive treatment always delays, and occasionally prevents the appearance of the sero-reaction, and this is likewise Gougerot's opinion who has seen the secondary manifestations delayed so long that they only appeared seven months after the chancre. Moutot and Lévy-Bing have besides noted numerous unsuccessful results from the abortive treatment. For my part, regardless of the rather considerable number of cases which have passed through my hands in Professor Oltramare's clinic, I opine to the belief that the question is yet too recent as regards the frequency of syphilitic relapses long delayed to form an exact opinion on this point.

It is not only on the question of abortive treatment that observers disagree; the same differences of opinion are encountered when the treatment of syphilis taken in its *ensemble*, is brought up. The publications on this subject are so extraordinarily numerous that it would be materially impossible to be complete, so that I shall quote some



writers selected at random. In 1908, Behring wrote that in the conduct of antisyphilitic treatment, the physician should endeavor to prevent the appearance of a positive reaction and should this be already present, it should be his duty to render it negative. Plehn has often seen a negative Wassermann coincide with active manifestations or, on the contrary, a positive reaction has persisted while the patient did not present a single specific lesion. In the latter circumstance Plehn is of the opinion that one must not be inclined to continue the treatment too long.

Letterer, on the contrary, employs Wassermann's reaction as a guide to treatment, believing that the sero-reaction is the only means at our disposal capable of controlling the syphilitic manifestations. Dreyer declares that the reaction is devoid of any value in respect to treatment and he relates a case in which the Wassermann remained negative for four years, although the patient was a victim to specific accidents. Nicolas and Charlet believe that the Wassermann is a therapeutic index of little value. Bruhns and Halbestaetter think that during the early years of the infection the disappearance of the reaction and absence of clinical manifestations are not sufficient to cause treatment to be stopped; in the late phases, on the contrary, a positive reaction is not an indication for resorting to treatment and that it is enough to watch the patient. Boas has remarked that the appearance of a positive Wassermann is often the index of an approaching relapse; therefore, immediate treatment should be given. According to Emery and Netter, the sero-reaction is a good means of controlling the treatment, because it frequently remains positive until all the lairs of the treponema have been extinguished.

In 1910, Gaston and Girauld stated that "a positive sero-reaction should indicate treatment, a negative reaction should not prevent treatment if lesions are present. . . ." Gaucher is opposed to the pretensions of those who believe that a syphilis is cured when the patient is free from specific manifestations and has a negative Wassermann. In 1912, Gougerot stated that the results of the sero-reaction alone were not enough to guide the physician in the treatment of the patient and in the same year Leredde and Rubinstein remarked that Wassermann's reaction "allowed one to control the results of treatment and no longer to blindly treat syphilitics." Finally, Lévy-Bing, Dogny and Gerbay, in June, 1914, after an exhaustive study

of the subject, concluded that "it is not possible to affirm that one can be guided by the results of sero-reaction so far as beginning or stopping antisyphilitic treatment is concerned."

Personally, I believe that in the treatment of syphilis, as in all other affections, the physician should be above all wary of two things, namely, to base the treatment solely on laboratory results and to employ the same line of treatment indiscriminately to all cases. The syphilographer should be at the same time a good clinician, capable of judging the degree of infection of the patient, his biological peculiarities, his capacity of reaction and his peculiar sensibility in relation to antisyphilitic remedies. He then can act in consequence and, without neglecting data of unquestionable utility which the Wassermann reaction will give in the majority of cases, he will not take it into consideration to the exclusion of other diagnostic signs, but will interpret it in conjunction with the other data obtained from the anamnesis and clinical manifestations. The sero-reaction is not infallible and there are rare instances where a negative Wassermann coexists with a specific lesion. Therefore, it is well not to place an exaggerated confidence in it (see Cases XIII and XXXIV).

Consequently, I am of the opinion that it is impossible and that it would be bad to attempt to lay down any hard and fast rules in treatment. However, there are general facts that may be well to consider; there are some principles that are useful to follow, but when face to face with the patient, they suffer numerous exceptions.

The abortive treatment should be continued for a number of months or even several years, regardless of a persistent negative reaction. The physician should never be affirmative that a cure of syphilis has been obtained, but should watch the patient and the state of his serum. The same principle, that is to say the continuance of an energetic treatment regardless of a persisting negative reaction, should apply to cases of recent syphilis during the entire secondary phase.

Should the Wassermann remain positive in spite of all kinds of treatment (Cases VII to XI) and especially after an energetic mixed treatment, it will be useless to persist too long, because often in such cases the sero-reaction will become negative of itself after cessation of all therapeutic measures (Cases XXI, XXV and XLII).

The appearance of a positive reaction after one or several negative ones is an absolute indication for treatment, a positive Wassermann being often the forerunner of a relapse (Cases XLVII and XXXIII).

In the tertiary period and in latent syphilis a positive reaction is not an absolute indication for treatment. The physician must judge of the opportunity of therapeutic interference, above all taking the evolution of the disease into consideration.

Contrary to the opinion of Lévy-Bing, Dogny and Gerbay, I believe that Wassermann's sero-reaction is a good guide to treatment, on the condition, however, that it is considered entirely from a clinical point of view.

## PITUITARY POLYURIA (DIABETES INSIPIDUS)

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FROM the earliest clinical conceptions of diabetes mellitus, the condition had been attributed to a grave functional nervous state, a vague organic lesion of the base of the brain, or an idiopathic perversion of renal function. The prognosis based upon these several hypotheses has heretofore been absolutely unfavorable, and the innumerable forms of therapy since 1682, when Willis<sup>1</sup> differentiated it from diabetes mellitus, have been of no avail. In recent years, studies in clinical medicine have clarified the etiology of this syndrome, so that now an effective treatment has been established, placing this condition in the group of remediable diseases. The striking therapeutic effects of the administration of extract of the posterior (and intermediate) lobe of the hypophysis, and the almost constant association of the syndrome of diabetes insipidus with other hormonal signs of pituitary neoplasm or disturbed pituitary function, has inclined the writers to consider a disordered hypophysis the cause of this symptom complex. In cases personally observed, the association of pituitary tumor or signs of disturbed function of some lobe of the hypophysis with polyuria has been so constant that it has been deemed not out of place to term the condition "pituitary polyuria," in order to more readily appreciate the connection of this syndrome with this endocrine gland.

The incidence of the disease is comparatively rare. Eichorst<sup>2</sup> saw but 17 cases out of 35,942, patients in the Medical Clinic at Zurich, between 1876 and 1897. Gerhardt<sup>3</sup> found 55 cases among 113,600 treated at the Charit , Berlin, from 1877 to 1896; Fletcher,<sup>4</sup> 7 cases out of 403,505 cases treated in the Johns Hopkins Hospital and Dispensary, from 1899 to 1904; the total averaging but 14 cases per 100,000.<sup>5</sup> The 11 cases herewith reported were present in a series of approximately 1000 patients showing definite clinical evidence of endocrine disturbance. Stoermer<sup>6</sup> found that the disease

occurred in men more commonly than in women; and Kulz,<sup>7</sup> in boys more frequently than in girls. The familial character has been stated by Clay,<sup>8</sup> Gee,<sup>9</sup> Lancereaux,<sup>10</sup> and Weil,<sup>11</sup> the last named reporting 17 cases in one family in three generations. In the series reported by the writers there are 10 men and 1 woman, the age incidence being between fourteen and thirty-five; with no suggestion of familial tendency.

The development of the pathogenesis of this dramatic clinical syndrome has been one of the most fascinating studies in medicine. In 1682, Thomas Willis<sup>1</sup> noted that the urine of some diabetic patients was and that of others not sweet, which led him to introduce the classification of saccharine and non-saccharine diabetes. In 1794, Johann Frank<sup>12</sup> defined the condition as diabetes insipidus, or spurious diabetes, as distinguished from diabetes mellitus, or true diabetes. Since this time there have been many theories, and a tremendous amount of literature accumulated regarding the causation and pathology of this disease. Three commonly propounded theories are (1) that the disease is due to faulty concentration of urine on the part of the kidneys; (2) that it is a primary polydipsia, with normal renal function; and (3) that it is a symptomatic polyuria induced by stimulation of the kidney from different causes: the older theories being of functional and organic nervous lesions, the more notable and recent pathogenesis being due to a disorder of hypophyseal secretion. There has, however, been no definitely consistent pathology of the disease established. Talquist,<sup>13</sup> E. Meyer,<sup>14</sup> Seiler,<sup>15</sup> and Socin,<sup>16</sup> thought the condition due to lack of ability of the kidney to secrete a concentrated urine. Finkelnberg,<sup>17</sup> Schwenkenbecher,<sup>18</sup> Falta,<sup>19</sup> Forschbach and Weber<sup>20</sup> did not believe the concentrating power of the kidney was lost, but that there was a pathological stimulation, which produced larger amounts. Spaether<sup>20</sup> considered diabetes insipidus to be a paresis of the renal vasomotor nerves, of either central or peripheral origin. Ebstein<sup>21</sup> was of the opinion that the condition was due to primary polydipsia, with resultant polyuria.

Although many cases of diabetes insipidus carefully studied showed no pathology, many others displayed definite lesions about the floor of the fourth ventricle or in the hypophysis. Claude Bernard,<sup>22</sup> in 1849, proved that by puncturing the medulla oblongata and the brain stem in various places, a definite polyuria would occur. In

1870, Eckhard <sup>28</sup> produced polyuria by various types of irritation of the vermiform process of the middle lobe, and injury to the posterior lobe of the brain. Kahler, <sup>24</sup> in 1886, produced a permanent polyuria and polydipsia in animals by injecting small quantities of concentrated silver nitrate solution into various portions of the brain.

In the course of these investigations, the hypophysis naturally demanded considerable attention. Schaefer <sup>25</sup> first demonstrated that the administration of posterior lobe extracts produced a marked polyuria in animals. Schaefer and Oliver, <sup>26</sup> and Magnus <sup>27</sup> and Herring <sup>28</sup> later confirmed this fact. E. Frank <sup>29</sup> and Cushing <sup>30</sup> noted the frequent association of the condition with lesions of the base of the brain particularly involving the hypophysis. Schaefer found a polyuria due to non-bloody mechanical or chemical irritation of the hypophysis. Crowe, Cushing, and Homans <sup>31</sup> noted the presence of a polyuria after operations upon the pituitary gland in animals, and the same occurrence and disappearance of polyuria following operations upon hypophyseal tumors in man. These clinical and experimental facts led to the conclusion that diabetes insipidus was due to a hyperfunction of the posterior and intermediate lobes of the hypophysis. This opinion obtained for a time, although many discrepancies were noted, both clinical and experimental, and the trend of opinion began to swing from the hypersecretory hypothesis to hyposecretion of the posterior and intermediate lobes of the pituitary.

The first significant work, in support of the latter view, was done by Pentimalli and Quercia, <sup>32</sup> in 1912, who found, in contradiction of Schaefer's work, that in the perfused, isolated kidney of the rabbit, extract of the posterior lobe caused *constriction* of the renal vessels, and that diuresis was diminished instead of increased. In 1913, Farini <sup>33</sup> and von der Velden <sup>34</sup> published the fact that extract from the hypophysis would decrease the amount of urine in cases of diabetes insipidus, as well as in healthy individuals. In 1914, Motzfeldt <sup>35</sup> demonstrated the same fact by the injection of Pituglandol (extract from the posterior lobe and pars intermedia,) and was of the opinion that the original experiments of Magnus and Schaefer <sup>27</sup> were faulty, due to the fact that they had used anesthetized animals. Motzfeldt, in his own work used rabbits without anesthesia. Polyuria would follow the introduction of water into the stomach of these rabbits and could be absolutely controlled and decreased by the sub-

outaneous injection of posterior lobe extracts. This effect was constant, and seemed to be independent of intestinal absorption and vagal influence. It could be prevented by section of the splanchnics or of the renal nerves near the hilus of the kidney. He concluded that the secretion of the pars intermedia exerted its antidiuretic effect in the stimulation of the sympathetic nerves, influencing especially the renal vasomotor system. He reported 3 cases in which there was nothing abnormal found in the pituitary, but a number of symptoms pointed to an insufficiency of the posterior lobe. Bab<sup>36</sup> believed the hypophysis a regulator for the control of the physiological functions of the urogenital system, and considered diabetes insipidus as a hypofunction of the pars intermedia, or a disturbance of the reception of the secretion in the pars posterior, or a propagation of the secretion in the cerebral lecnæ. Many clinical discrepancies had been noted; for example, as remarked by Biedl,<sup>37</sup> the association of diabetes insipidus with dystrophia adiposogenitalis. These considerations led to a reversal of opinion, the consensus being that diabetes insipidus was due to hyposecretion of the pars intermedia and pars posterior. There have been multiple reports following, dealing largely with the therapeutic effect of various forms of pituitary extract upon the polyuria: Roemer<sup>38</sup> (1913 and 1914), Biach<sup>39</sup> (1914), Frey and Kumpies<sup>40</sup> (1914), Umber<sup>41</sup> (1914), Falta<sup>42</sup> (1915), E. Graul<sup>43</sup> (1915), Hoppe-Seyler<sup>44</sup> (1915), Kleeblatt<sup>45</sup> (1915), von Konschegg and Schuster<sup>46</sup> (1915), Strauss<sup>47</sup> (1916), Bab<sup>36</sup> (1916 and 1917), G. Maranon<sup>48</sup> (1917). Barker and Mosenthal,<sup>49</sup> in 1917, reported 2 cases in which the polyuria was markedly controlled by the injection of Pituitrin and Pituitary Liquid. They state that the urine was diminished in amount, the specific gravity increased, and the percentage of sodium chlorides and nitrogen content increased by the injection of extract of the pars posterior and pars intermedia. Tethelin (anterior lobe extract) and Adrenalin were of no value. They believe the kidneys to be healthy in diabetes insipidus, and that the condition is one of deficiency of secretion of the pars intermedia. Barker suggests the possibility that substances closely allied to Pituitrin, such as Histamin, might be substituted. The similarity of Histamin and pituitary extract in their action upon the uteri of mice and guinea-pigs, has been recently expressed by Abel and Macht;<sup>50</sup> while Cow<sup>51</sup> finds opposite effects, leading him to conclude that the plain

muscle stimulating principle of Histamin and of posterior lobe are not identical, Jackson and Mills<sup>52</sup> likewise find differences.

In addition to the theories concerning hyper- and hyposecretion of the hypophysis cerebri in diabetes insipidus, other investigators have advanced the theory that the hypophysis is not necessarily involved, maintaining that lesions elsewhere about the floor of the fourth ventricle, or in the midbrain, may be productive of the syndrome. The earlier experiments of Claude Bernard,<sup>22</sup> Eckhard,<sup>23</sup> and Kahler,<sup>24</sup> quoted previously, would seem to support this opinion. Houssey<sup>53</sup> does not accept the view that the polyuria is due to a diuretic hypersecretion of the pituitary gland, and states that in his experience the pharmacological action of pituitary extract was so variable that it was not permissible to deduce an insufficiency of the pituitary body from the successful use of the extract in controlling polyuria. He asserts that different immediate effects occur when pituitary extracts are injected intravenously, and that the chemical composition of the gland is very constant, as are the physiological effects of the gland extract, but that these effects vary qualitatively according to the methods of preparation. He states there occur in the pituitary extracts both renoc Contractor and renodilator substances, one or the other predominating, according to the circumstances, the *diuretic* effect running *parallel* to the *renovascular* effect. He disagrees with Cushing in his claim that the cerebrospinal fluid produces the same effect as pituitary extract, or that the secretion is poured into the fourth ventricle, as affirmed by Herring. His animal experiments demonstrated that the removal of the hypophysis produced decided differences in adult and very young dogs. In the adult an oliguria often occurred, while in the puppy polyuria was a constant finding for a few days succeeding operation. He believes that both these changes in urinary output were due to trauma, as these same changes in diuresis were observed in those animals in which the hypophysis was entirely removed. He studied in detail trauma to the neighborhood cerebrobasal zone and the pituitary body itself, by employing iron filings, it being possible, by means of the X-ray, to observe the effects upon dogs for a long time. Injuries within the determined zone produced polyuria. Injuries without the zone did not result in polyuria. He believes the polyuria was not due to pituitary involvement, but that it was produced by injury to the neighborhood tissues in close



proximity to this gland. In one case he punctured the infundibulum and penetrated into the third ventricle, without injuring the brain and without developing a polyuria. He defines the zone within which injury is productive of polyuria as limited behind by the peduncle protuberances, anteriorly by a line near the anterior limit of the optic chiasm, and laterally by the hippocampal gyrus. He does not believe that the pituitary constitutes a part of this zone, although it may be possible that the posterior lobe does participate.

Leschke,<sup>54</sup> while admitting that lesions of the hypophysis, such as trauma, sarcoma, carcinoma, tuberculosis, syphilis, etc., have been demonstrated in cases of diabetes insipidus, similar lesions in the mid-brain, especially those involving the tuber cinereum, may produce diabetes insipidus, with the hypophysis intact. He regards the hypophyseal lesion as purely incidental, and if associated with diabetes insipidus, deems the latter condition due to pressure or extension of the lesion into the tuber cinereum. J. Camus and G. Roussy<sup>55</sup> contend that experimental data prove that lesions of the tuber cinereum and infundibulum are productive of polyuria. They found that after removing the hypophysis in dogs, pituitary treatment did not improve the syndrome of diabetes insipidus, from which fact it was inferred that the essential factor might be a lesion of the adjacent zone of the brain. Claude and Lhermitte<sup>56</sup> described a case presenting bitemporal hemianopsia, attacks of dysarthria, amnesia and delirium, attacks of narcolepsy, tachycardia, irregularity of the heart beat, extra systoles, embryocardia, increased blood-pressure, polyuria, and polydipsia, in which post-mortem examination disclosed a tumor formed from the ventral wall of the third ventricle and distinctly confined to the *infundibulum*, stating that the hypophysis was completely normal. This article attempts to ascribe polyuria accompanied by polydipsia to lesions of the base of the third ventricle, and not to lesions of the hypophysis. Olæches (M.)<sup>57</sup> reports a case, leading him to subscribe to the infundibular hypothesis. On the other hand, Luzzato (A. M.)<sup>58</sup> contends that destructive lesions of the neurohypophysis or pedicle may produce the syndrome, with no demonstrable lesion of the infundibulum.

Chiasserini<sup>59</sup> found that hyperplastic changes in the *pars intermedia*, experimentally produced by injections of sporotrichia

**FIG. 1.**



**Case 1.** Note eunuchoid type, with over-development of long bones; long hands, with slender, tapering fingers.

100

100

and tubercle bacilli into the sella turcica or hypophysis, resulted in polyuria.

Thus it may be seen that the accumulated data to date, independently of such considerations as local kidney conditions or primary polydipsia, offers a considerable variety of causation: (1) Hypersecretion of the posterior (pars nervosa) and intermediate (pars intermedia) lobes of the hypophysis. (2) Hyposecretion of the posterior and intermediate lobes of the hypophysis. (3) Disorder of the intermediate lobe (pars intermedia), without involvement of the anterior lobe or the pars nervosa of the pituitary. (4) Lesions of the hypophyseal neighborhood, without involvement of the hypophysis proper. It must be accepted, as Simonds<sup>22</sup> pointed out, that lesions occur in the specified zones without polyuria, and on the other hand, the syndrome of diabetes insipidus may exist without demonstrable lesions of the hypophysis or hypophyseal neighborhood. Yet it must be borne in mind that the *histological knowledge of the hypophysis is so indefinite that no deductions can be based upon it relative to the activity of the secretions or functions of the individual lobes of this endocrine gland.*

#### REPORT OF CASES

CASE I.—F. B., m., twenty-eight, Gen. No. 1203.

*History.*—First symptoms began about a year and one-half previous to first observation, with general nervousness, inability to sleep, and chilly sensations. Nervousness became more marked, insomnia marked, patient being unable to apply himself or retain anything he would try to read, during following eighteen months. Increased libido was noted by frequent nocturnal emissions. Constipation had been present for six or seven years, associated with nausea and occasional vomiting, distention of the abdomen with gas. At the age of twelve, apolyuria, albuminuria and glycosuria were found. Patient was also troubled with biliousness, cramps in the abdomen, and occasional fever. Until the age of six he was healthy, of normal weight. From the ages of six to ten he was thin, felt well. From ten to fourteen he was sick most of the time, complaining of weakness, nervousness, loss of appetite, biliousness, boils, skin eruptions, cramping in the abdomen, diarrhoea, and occasional fever. He recovered from these symptoms at age of sixteen, and from that age to twenty

was in good health, of normal weight and height. At the age of twenty he began losing weight, but felt fairly well. Nervousness was manifested only after four or five years. Since the age of twenty he has been under weight, and for the last two or three years he has not felt well. All the above complaints, including *polyuria*, severe *headaches*, and *vomiting*, have been increased during the last two or three years. *Past*: Measles, mumps at the age of eleven (no orchitis). *Personal and family*: Negative.

*Examination.—General*: Lower measurement, 38 in. (96 cm.); upper, 34½ in. (87½ cm.); span, 78 in. (198 cm.). Tall, slender, eunuchoid type of individual. Very slight growth of hair about thighs and upper portions of legs, lower legs comparatively free. Absolutely no abnormal hair growth about upper portion of body. Hands very long, palms narrow, fingers long and tapering, lunular markings visible. Distance from wrist-joint to tip of middle finger, 22 cm.; from knuckle to tip of middle finger, 12 cm.; circumference of palm at widest portion, 22 cm.; length of arm from elbow to lower end of radius, 30 cm. Feet of same general character as hands, very long. Size of shoe, 10½, medium last. Faint brownish pigmentation about forehead, extending down on lateral aspects of cheeks and neck. No scars or lesions about body except brownish scar on right anterior leg (old infection following bruise). Skin of body warm, with exception of feet. Pulse slow, regular, 60–80. Blood-pressure, 120/85. Temperature constantly subnormal, 96 to 98. *Regional*: Negative with exception of proportionate overgrowth of all bones. *Urine* (see Fig. 2): 3000–4800 c.c., sp. gr., 1000–1010, free from albumin, sugar and morphological elements. *Blood* (including Wassermann): Normal. *Carbohydrate tolerance* normal: Blood sugar after fifteen hours' fast, 0.13 per cent. (normal, 0.10–0.12 per cent.); one hour after 123 gm. dextrose, 0.156 per cent. (normal, 0.16 per cent.); two hours after same, 0.112 per cent. (normal 0.10–0.13 per cent.). *Basal Metabolism*: –3 per cent. *Stomach analysis* (Ewald breakfast): Free HCl 15, combined acidity 7, total acidity 22, lactic acid negative, microscopic normal. *X-ray Sella turcica* small, anteroposterior diameter 10 mm., depth 5 mm. *Fluoroscopic of stomach and colon* normal. *Röntgenogram of chest* negative. *Röntgenogram of hand* shows no definite tufting of exostoses, slight overgrowth of metacarpophalangeal bones.

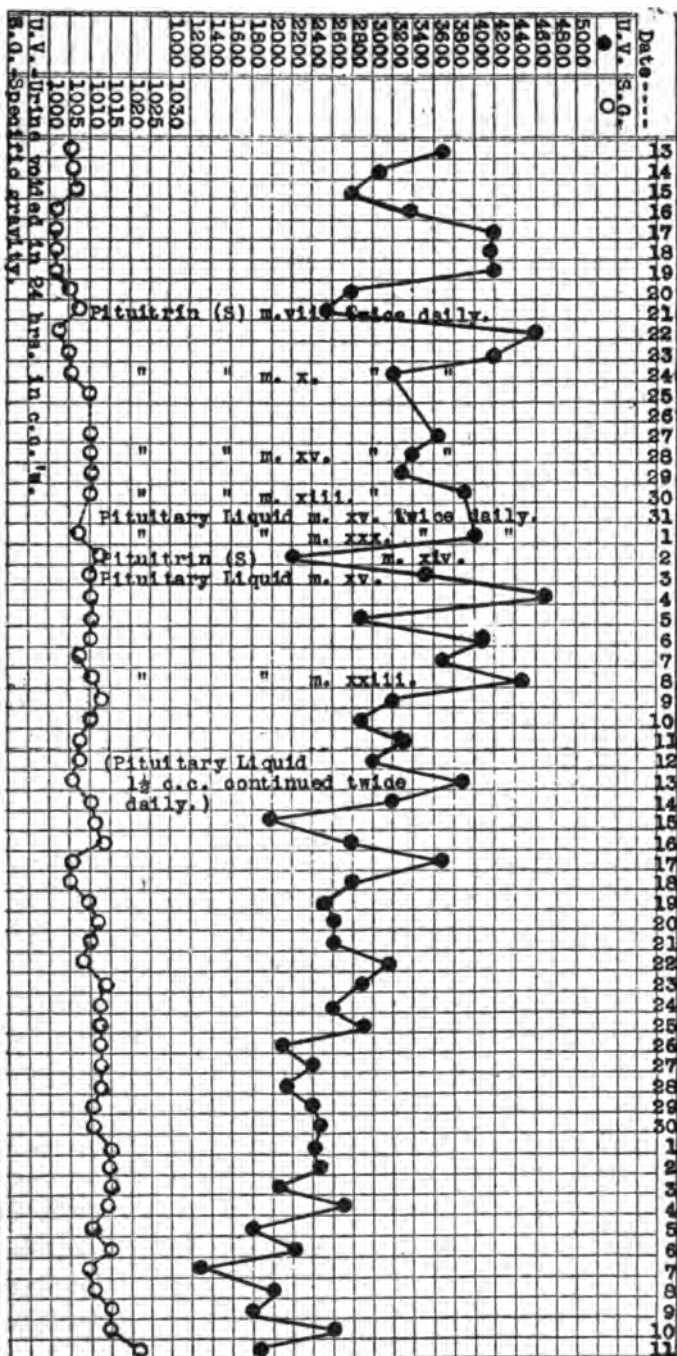


FIG. 2. CASE I.

*Results of Treatment.*—(See Fig. 2.) Entered St. John's Hospital October 12th and was dismissed December 12, 1918, during which time he was given Pituitary Substance, grs. x, after meals, and increasing dose of Pituitary Liquid, from m, vii to m. xxx, twice daily. Total amount of urine dropped from 4800 to 1600–2000, specific gravity increased from 1000 to 1015–1020. Headaches, nausea, vomiting and muscles fatigue disappeared entirely. Patient was then dismissed from the hospital and placed upon Pituitary Substance (Entire Gland), gr. v t.i.d. p.c. During the following six months there was no recurrence of polyuria, polydipsia, polyphagia, nervous or gastric symptoms. For the following one and one-half years discontinued all treatment with only an occasional recurrence, particularly when under tension or strain, at which time he would have a transient polyuria, polyphagia and polydipsia for a few days, associated with nervousness and fatigue. At this time was again placed upon Pituitary Substance, grs. x, t.i.d., and a few hypodermics of Pituitrin which controlled the tendency to transient relapses, even when working under tension, and improved his general condition.

CASE II.—C. K., m., twenty-three, Gen. No. 911

*History.*—At the age of ten the patient became conscious of passing abnormal quantities of very light-colored urine. There was increased appetite, polyphagia, with preference for carbohydrate foods, without marked polydipsia. Constipation was extreme at that time. Susceptibility to respiratory tract infections was decided. Urticarial skin eruptions and labial herpes were frequent. The patient was remarkably susceptible to chilly weather. Periodical diarrhoeas, of one or two days' duration, followed by obstipation, were of frequent occurrence. There was perverted appetite for condiments, such as mustard, pepper, ketchup, etc. At the age of eleven the teeth softened, requiring constant attention. General nervousness and irritability was a distressing complaint. The patient was easily annoyed, introspective, apprehensive, and easily startled. At that time he began having mild gastric disturbance, sense of weight, fullness and eructations, associated with cardiac palpitation and general vagotonic disturbance, coming on immediately after meals. At about the age of fourteen rapid growth occurred, confined entirely to height, without increased obesity. The height had a tendency to the long-bone variety. Slight dyspnoea, not a distinct asthma, was manifested occa-

sionally, never bearing relationship to exertion. During this entire period there was steady increase in urinary output. The appetite became more ravenous, and the patient began to manifest evidence of abnormal thirst and liquid intake. There was profuse increase in discharge of mucus from the nose and mouth. From the age of ten to fifteen, he had frequent headache, always frontal in variety. At that time he became aware of marked lassitude, beginning disinclination for effort, either mental or physical. The symptoms gradually increased for the next two years. Puberty was retarded, development of secondary sexual characteristics occurring at the age of seventeen. Smoking improved the general symptomatology. Following adolescence, all symptoms were gradually intensified, with increased nervousness, irritability, depression, lack of confidence, memory defect, introspection, increased gastric disturbance, and tendency to polydipsia, polyphagia, and polyuria. If he missed breakfast, he would be miserable until the noon meal. Two years previous to observation, while serving in the army in the Northern woods (aeroplane production department), the patient gained 30 lb. in weight, attributed partially to the use of mechanical influence upon the bowels, such as bran, etc., but with this increase in weight there was no change in lassitude or increase in energy. This weight was retained for a year, after which time there was decided increase in the symptomatology and rapid loss of weight—30 lb. in two months. Lethargy was increased, with marked drowsiness and somnolence; tendency to lie down if the opportunity afforded. Cramping pains in the extremities and scattered areas of anæsthesia were present. There was a return of sensitiveness to cold. Intensification of the auditory disturbance occurred in the form of musical notes constantly repeated, which seemed to be projected externally. The entire picture during the last year has been a gradual definite increase in the entire presentation, the principal manifestations being headache, auditory disturbance, visual disturbance (consisting of misting and general diminution of acuity, with bitemporal tendency), increased tenaciousness of salivary mucus, dyspnoea (improved by exertion), general nervousness, spasmodic jerking, irritability, apprehension, depression, cardiac palpitation associated with gastric disturbance, introspection, etc. Polydipsia, polyphagia, and polyuria have been intensified, the polyphagia being particularly manifest. The patient has a ravenous



appetite, eating abnormal quantities of food, which does not satisfy his hunger, and continually has a tendency to procure food between meals, the temptation to buy fruits in large quantities being impossible to resist. There is strong preference for such foods as cake, ice-cream, etc. The above complex has a psychological aspect, inasmuch as when the patient is alone his condition is decidedly decreased, intensified in the presence of others. Libido dates back about to the time of development of the secondary sexual characteristics, and has undergone constant definite decrease, until there is practically complete impotence. Libido and nocturnal emissions were intensified at the age of seventeen by nicotine. *Past*: Scarlet fever at the age of two. Frequent colds. *Personal*: Habits of life fairly regular. No tobacco at the present time. Alcohol for a period of two months (at the age of twenty-one), otherwise negative. Tea and coffee moderately. *Family*: Father living, fifty-five, tall and thin in stature, not well. Mother living, fifty, small and thin; not well for the past ten years, highly energetic. One brother not mentally balanced, without being definitely insane. One sister, inclined to obesity. Grandfather on paternal side was mentally unbalanced.

*Examination.—General*: Lower measurement, 35 in. (89 cm.); upper, 33½ in. (85 cm.); span, 74 in. (188 cm.). General development, tall, eunuchoid individual. Hands fairly broad, fingers not abnormally long, some tendency to tapering. Feet of same general character. Slight abnormal distribution of hair about lower portion of body, none about upper. Yellowish pallor across abdomen, sclerae very slightly tinged, but not jaundiced. Blood-pressure, 110/75. Pulse, 60. Temperature constantly subnormal, 96° to 98°. *Regional examination* reveals no positive abnormal signs. *Urine*: Previous to treatment, 4500–4800 c.c., sp. gr., 1008–1010, clear, free from albumin, sugar and microscopic findings. *Carbohydrate tolerance* increased: Blood sugar fifteen hours' after fast, 0.09 per cent. (normal, 0.10–0.13 per cent.); one hour after ingestion of 1.59 gm. dextrose per kilogram of body weight, 0.096 per cent. (normal, 0.18 per cent.); second hour after same, 0.093 per cent. (normal, 0.15 per cent.). *Sputum*, negative. *Blood* (including Wassermann): Normal. *Pituitrin reaction*: Intestinal reaction occurred following injection of 36 minims of Pituitary Liquid (Armour & Co.). *Röntgenogram of chest*, normal. *X-ray of Sella turcica* small, antero-posterior

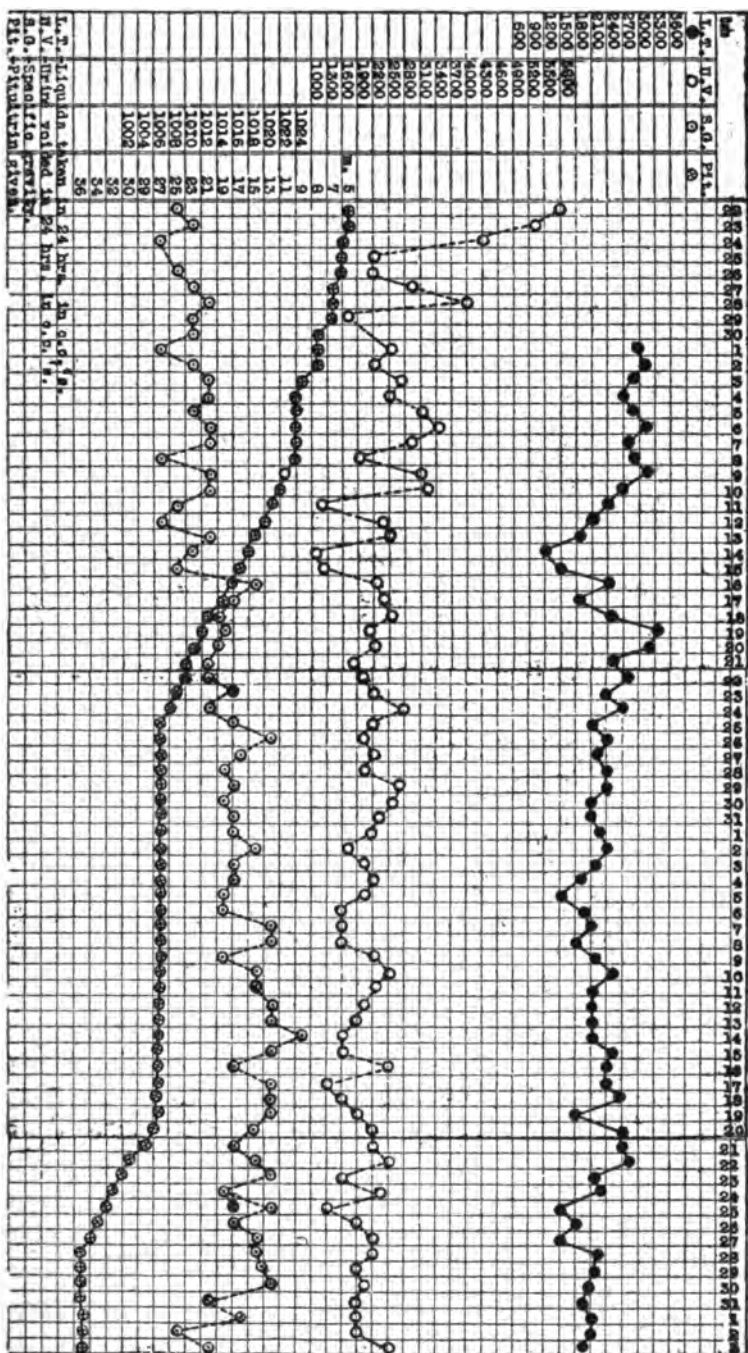


Fig. 4. Case II.

diameter, 10 mm.; depth, 8 mm. *Fluoroscopic examination of stomach and colon*, negative.

*Results of Treatment.*—As shown by the chart, there was marked reduction of polyuria and increase in specific gravity of the urine. Polydipsia and polyphagia were favorably affected to the same degree. The patient's general weakness, nervous depression, and melancholia, simulating maniac depression, were greatly improved while under hospital observation.

CASE III.—A. H. S., m., thirty-one, Gen. No. 1115. Referred by Dr. W. W. Graves, St. Louis.

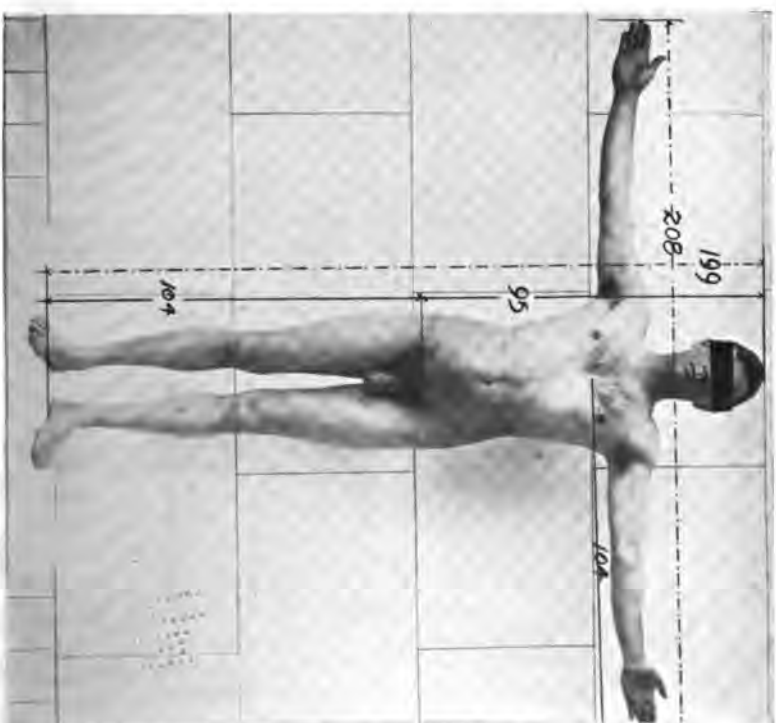
*History.*—Following operation for hernia one and one-half years previous to first observation, onset occurred with physical exhaustion. Prior to this experienced pain in the right sacro-iliac region. Following operation has been unable to do a full day's work. Recently even short walks causing exhaustion. Associated with this general feeling of weakness, there is at times mental confusion as to purpose, indecision as to what course to follow. The chief complaint is frequency of urination, the amount of urine passed in twenty-four hours being two-thirds gallon. Polydipsia was marked, "could drink a gallon of water at one time." Other symptoms were headaches, particularly over the left eye (sometimes over both), and in the temporal regions, with bitemporal tenderness. Accompanying these headaches there is visual disturbance, scotomata, but no diplopia or hemianopsia. Roaring, ringing, and buzzing in the ears have been noticed since the onset. Shooting pains occur in the nose, from the glabella to the tip, and the nose is increasing in width. Increase in size of the face has been noted, particularly above the eyes, the cheek bones, and the lower jaw. The face swells at times, and there is increased pigmentation, particularly about the eyes. The teeth have been separating during the last year, more noticeably in the lower jaw. Previous to that time there had been no separation, except of the central incisors, both above and below. The tongue feels somewhat thick, the voice is changing. There is some difficulty in articulation, producing stuttering and stammering. Tickling and sensation of fulness in the throat are frequent. Gustatory disturbances, such as food and water tasting sweet, and lemons not so bitter and sour as normal, occurred at times. The sense of smell was also perverted, things smell "warm and gassy." Polyphagia was present

FIG. 3.



Case II. Note same general type of Case I, overgrowth of long bones and abnormal distribution of pubic hair.

FIG. 6.



Case III. Note gigantism, due to preadolescent anterior lobe hyperpituitarism, and secondary tendency to acromegaly denoting persistence of hyperactivity of anterior lobe in postadolescence.

FIG. 6.



Case III. Note separation of both upper and lower central incisors (Grave's sign).

FIG. 7.



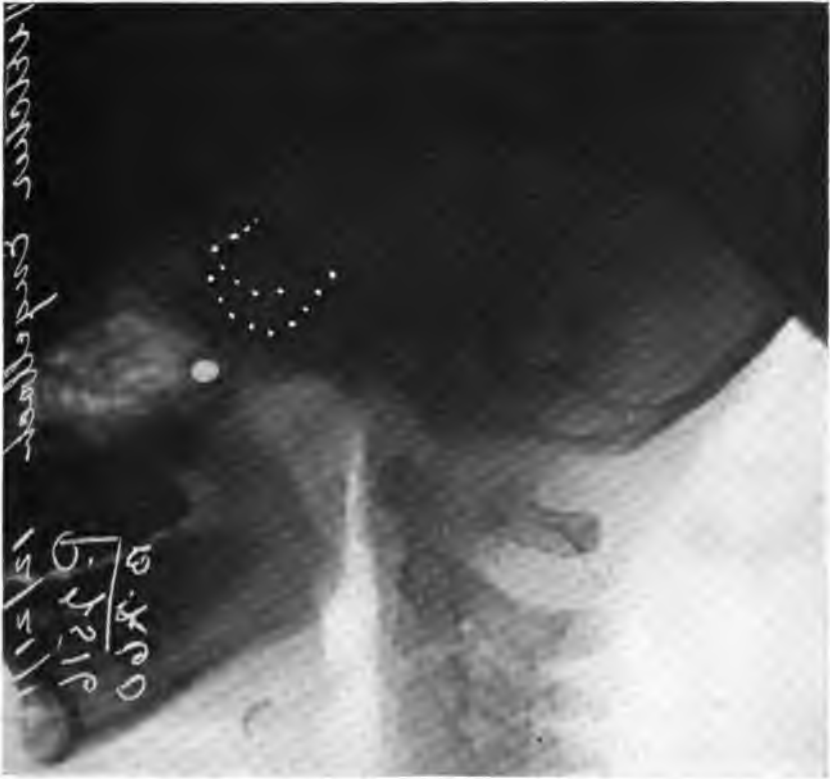
Case III. Note broadness of wrist and palm, and shortness of fingers, with slight blunting (tendency to acromegalic type).

FIG. 8.



Case V. Note extreme height, unusual length of long bones, long distance from symphysis to soles of feet, absence of obesity, and presence of well developed genitalia.

FIG. 9.



Case V. Röntgenogram of head. Note marked enlargement of sella turcica.



**FIG. 10.**



**Case VI.** Note well developed long bones; absence of adiposity; well developed genitalia; absence of hair on torso and extremities; normal-sized head, nose, chin, hands, and feet; narrow hips and genu valgum; and abnormal distribution of pubic hair.

and progressive. He gained 12 or 15 lb. in weight during the last three months, the present weight being 225 lb., height 6 ft. 7½ in. At the age of fifteen he was 5 ft. 8 in. tall; between the ages of fifteen and twenty-two, he grew about 12 in., continuing to grow until the age of twenty-four. He has grown about ½ in. during the last five or six years. He complained of aching in the extremities, particularly of the muscles in the upper arm and upper leg. The hands and feet swelled at times, and definitely increased in size in the last year. There has been some loss of libido for about one and one-half years, previous to which time sexual power was normal. Vague pains in the abdomen were present, with distention, belching, and some constipation. Intermittent pruritus over the body was distressing. *Past:* Measles, mumps, chicken-pox, malaria, bronchitis. Herniotomy as above. Tonsillectomy time of herniotomy. *Personal:* Appetite increased. Bowels irregular. Patient sleeps poorly (only two or three hours at night). Smokes twenty pipes of tobacco a day. *Family:* One brother is 6 ft. 3 in. tall, weighs 190 lb., well. Father is 6 ft. 3 in. tall, well. Mother is 5 ft. 11 in. in height, weighs 185 lb., well. Two sisters, well, one 5 ft. 10½ in. tall, weighing about 300 lb.; the other 5 ft. 8 in. tall, weighing 185 lb. Tendency to tall and large stature on father's side of family, and similar tendency on mother's side. Mother had "spasms" (losing consciousness) for four or five years, at about the age of forty-five.

*Examination.—General:* Upper measurement, 37½ in. (95¼ cm.); lower, 41 in. (104 cm.); span, 82 in. (208¼ cm). Circumference measurements: forehead, 31 in. (78¾ cm.); upper maxillæ, 21¼ in. (54 cm.); chin, 21½ in. (54½ cm.); neck, 16¼ in. (41¼ cm.); nipple, 39½ in. (100¼ cm.); navel, 35½ in. (90 cm.); hips, 41¼ in. (104¾ cm.); middle thigh, 21 in. (53¼ cm.); leg, 14½ in. (36¼ cm.). Size of shoe, 12 EE; of glove, 11. Wrist circumference, 7½ in. (19 cm.). Mixed type of gigantism, with secondary acromegalic signs. Marked separation of upper and lower teeth. Very slight chloasmic pigmentation. Slight abnormal distribution of hair across chest and nipples, and on mid-abdomen, slightly above umbilicus. Genitalia well developed, testicles 2 x 1½ in., penis 5¼ in. Musculature, hypertrophic throughout. Hands large, unusually well proportioned, about third larger than normal hand. No marked widening of tips of fingers, lunular markings present.



Feet of same general characteristics. Pulse normal. Blood-pressure, 119/80 (right arm); 119/70 (left arm). *Regional* examination negative, except for local findings of gigantism, pertaining particularly to osseous system. Endocrinous heart. *Urine* (twenty-four-hour specimen), 5500 c.c., clear, sp. gr. 1010, trace of albumin, sugar negative, microscopic negative. *Blood* (including Wassermann): Normal. *Carbohydrate tolerance* increased: Blood sugar after fifteen hours' fast, 0.087 per cent. (normal, 0.10–0.13 per cent.); one hour after 165 gm. dextrose, 0.131 per cent. (normal, 0.18 per cent.); two hours after same, 0.108 per cent. (normal, 0.15 per cent.). *Sella turcica*: Antero-posterior diameter, 15 mm.; depth, 15 mm. (slightly enlarged, but not considered abnormal). *X-ray of hand*: Tufting of distal phalanges, bones unusually large and dense. *Electrocardiogram*: Premature ventricular contraction, flattening of "T" wave and split "P" in Lead 3. *Basal metabolism*: First observation, —1 per cent.; one month after treatment (anterior and posterior lobe pituitary gland), +21 per cent.; eight months later (treatment had been interrupted in meantime) +1.7 per cent.; one month later (anterior lobe treatment given alone, posterior lobe discontinued), —7.5 per cent.

*Results of Treatment* (consisting of substances from both lobes of the pituitary gland).—Antuitrin and pituitrin hypodermically and pituitary substance by mouth had an ameliorating effect, decreasing the polyuria and increasing the libido. No decided results were noted in the muscle fatigue, headaches, general disability, or incapacity.

CASE IV.—C. W. F., m., thirty-two, Gen. No. 1194.

*History*.—Attention was first attracted to the patient's condition four years ago by a life insurance examination, in which the urine was found to have an abnormally low specific gravity. The specific gravity was 1004, verified by repeated examinations. During the next year and one-half no feature of interest occurred except continuance of low specific gravity (without polyuria). Following this he began to pass larger amounts of urine, ranging around 2500 to 3000 c.c. Nervousness, always present to a degree, was somewhat exaggerated, with greater tension and tendency to irritability and worry. There was no associated polydipsia, except occasional increased thirst; absolutely no polyphagia. Two and one-half years

later headaches commenced, usually bitemporal, frontal, and in anterior vertex, about the coronary suture, as a rule localized with greatest intensity in a line drawn from one temporal region to the other. These headaches were not associated with visual disturbances, as hemianopsia, or with nausea and vomiting. The patient usually awakens with headache, which persists throughout the entire day, not intensified by use of the eyes, physical effort, or other features of daily routine. They are present about half the time, averaging from two to three days a week, probably preventing his full efficiency. During the past three months there has been some increase in both frequency and severity of headache. Within the past three months the urinary output has been somewhat decreased, the general nervousness remaining about the same. Urine (twenty-four-hour specimen), 2500 c.c., cloudy, pale, sp. gr. 1005, total solids 29 1/10 gm., reaction acid; albumin, sugar, indican and blood negative, few hyaline and granular casts, few pus cells. Wassermann negative with two antigens. July 3, 1917: Urine, sp. gr. 1007, trace of indican, negative microscopically except for occasional leucocyte. September 19, 1918: Urine, sp. gr. 1015, acid, negative microscopically except for occasional pus cell. A diagnosis of diabetes insipidus was made by Drs. R. C. Harris and W. U. Kennedy (St. Louis). Strychnine administered by Doctor Harris was of no benefit, and the patient was put upon probable specific treatment by Dr. Henry Jacobson, without avail. Radiographs of the teeth were made showing a definite root abscess about the first lower molar, which was removed. *Personal*: Appetite variable. Patient sleeps well. Constipation intermittently. Three or six cigars daily for the last six years. *Past*: Mumps, whooping cough. Patient was struck on the head with a baseball bat fifteen years ago. Brief concussion. *Family*: Father living, 64, hypertension. Otherwise negative.

*Examination.—General*: Symphysis to soles of feet, 36¾ inches (93¼ cm.); symphysis to vertex, 32¼ inches (81¾ cm.); span, 75 inches (190½ cm.). General development, tall individual, with considerable emaciation, eunuchoid measurements. Slight growth of hair about legs and fore-arms, abnormal distribution of hair about upper portion of chest. Hands long and slender, fingers slightly tapering, lunular markings visible. Cyanosis of fingernails. Hands cold, no perspiration. Feet of same character. Color about body

slightly sallow, very good about face. Pulse, 60. Temperature, normal. Blood-pressure, 118/92. *Regional examination* normal. *Urine* varies from 3000 to 4000 c.c., light, sp. gr. 1010–1015, free from sugar, albumin, microscopic findings, etc. *Blood* (including Wassermann) normal. *Sugar tolerance* normal: Blood sugar after fifteen hours' fast, 0.105 per cent. (normal, 0.10–0.13 per cent.); one hour later and following ingestion of 99.3 gm. dextrose, 0.144 per cent. (normal, 0.18 per cent.); two hours after same, 0.096 per cent. (normal, 0.15 per cent.). *Basal metabolism*, 8 per cent. *Pituitrin intestinal reaction* produced by minims 12 of Pituitrin (0). *Sella turcica* small.

*Results of Treatment.*—Pituitrin treatment reduced the palyuria to normal and alleviated the general symptoms, such as general weakness, nervousness, and headache, almost completely. Interruption of treatment, however, would allow the patient to relapse into the same condition as previous to its institution.

CASE V.—J. N., m., fourteen, Gen. No. 748.\*

*History.*—At the age of ten the child fell about thirty feet, striking on the lower portion of the back, and was picked up unconscious. After recovery he got up and played around with the other children. Recovery from this fall seemed to be complete. Following this, the patient for some time passed *an unusual amount of urine*. One year previous to observation, while in school, the patient noticed white spots before the eyes and had to be escorted home. Following this, double vision occurred, and pains in the head appeared, gradually becoming worse, but was able to be up and around. He complained next of difficulty in urination. Walking became more difficult, vision was more affected, and finally, thirteen weeks previous to observation, the patient was confined to bed. Vision, pains in the head and neck, and vomiting (without nausea) became gradually worse. The diet has never been abundant, on account of the poverty of the parents. The patient was in Mullanphy Hospital, St. Louis, Mo., seven weeks previous to observation, where a diagnosis of brain tumor was made. He remained in the hospital twenty-four hours. After he returned home his condition became gradually worse, until his entrance to the present service. *Past:* Measles. *Family:* Negative. *Note:* History

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\* Reported in detail in *Medicine and Surgery*, Vol ii, No. 4, April, 1918.

indefinite on account of its source, through a friend of the patient. Patient too somnolent to answer questions.

*Examination.—General:* Height, 5 feet 5 inches. Marked emaciation, joints appear very much enlarged, due to muscles and soft tissues between joints being considerably atrophied. Bony markings throughout entire body very pronounced. Patient unable to stand alone, but able, by holding on, to bear weight on legs. Long bones overdeveloped, proportions eunuchoid. Genitalia normal size. No abnormal distribution of hair about body or extremities, no abnormal pigmentations or skin lesions. Hair on head, eyebrows, and lids very thick and well developed; none on face except very slight growth at angles of mouth. Color, nails very anæmic; skin colorless. Skin harsh, very slight desquamation over arms, none on malar processes of face. Dull, apathetic, stuporous, reacts to stimulus, answers questions imperfectly, complains of pain in back of head. Marked divergence of right eye. Retraction of abdomen, flaring of ilial regions, symphysis prominent, ribs showing through skin very distinctly. Temperature, 97.6°–99.2° F. Pulse, 86–120. Respiration, 16–28. Positive regional signs: *Eyes*, sight almost entirely gone, can distinguish light from darkness, not able to distinguish (count) fingers or recognize ordinary object such as lead pencil. Cannot distinguish colors. Marked divergence of right eye, also slight turning upward. Pupils dilated, right pupil slightly larger than left. Right eye will not turn further than median line to left. Pupils do not react to light or accommodation. Scleræ clear, no marked pigmentation of lids, lid movement normal. *Urine* practically normal, including amount. (Note history of *early polyuria*.) *Blood* (including Wassermann) negative. *Carbohydrate tolerance*: 100 gm. of glucose produced no glycosuria in twenty-four-hour specimen. *Röntgenogram*: Marked enlargement of sella turcica. *Ophthalmic*: Complete optic atrophy, complete loss of vision (perimeter chart impossible).

*Note.*—Patient died three weeks after leaving the hospital, from increased intracranial symptoms, cachexia and coma (autopsy refused).

*Diagnosis.*—Hypopituitarism, posterior lobe insufficiency without anterior lobe involvement: preadolescent variety, neoplastic type.

CASE VI.—H. B. T., m., thirty-five, Gen. No. 1622. Referred by Dr. Geo. Sihler, Jr., Litchfield, Ill.\*

*History.*—March 17, 1917. Eight years previous to first observation, following typhoid fever, the patient had a condition characterized by marked *polyuria* (nycturia), *polyphagia*, and *polydipsia*. This was associated with rapid, extreme emaciation, progressive anæmia, weakness, and marked general nervousness. The urine was repeatedly examined for sugar and found negative. The above group of symptoms continued for three years. Following this, there was a remission for three years, during which time there was *marked improvement* of all symptoms. The patient *gained weight*, the anæmia disappeared, and the general condition apparently returned to normal. During this time he noted slight enlargement of the fingers and increase in size of the hands and feet. Six years after the onset of the disease, while the patient was under a severe nervous and mental strain, there occurred the first evidence of the second exacerbation. This began with severe *emotional symptoms*, consisting of uncontrollable periods of crying. At that time extreme drowsiness and torpidity were first noted. Soon afterward the patient began to sleep continuously for twenty-four hours at a time, and it required constant effort for him to keep awake. Physical weakness, emaciation and anæmia recurred. Intermittent diarrhœa was present frequently throughout the third period of the course. An *abnormal appetite* developed, but there was no return of polydipsia or polyuria. Two years ago *headaches* were first observed. This headache was located high in the temporal region, radiating back over the parietal bones, down into the occipital region and upper cervical vertebræ. It was very severe in character, consisting of deep-seated throbbing pain, at times changing into tearing pain in the vertex of the skull. It was markedly intensified by the use of strychnine, becoming almost unbearable after the exhibition of this drug. Two months after the onset of headache, the *eye symptoms* commenced. The initial defects of vision began in the left eye, consisting of dimness of vision affecting principally the temporal side. This became progressively worse for a time, then began to improve, returning almost to normal, in the left eye. Soon after the vision in the left eye reached normal, the right eye became defective. This right eye visual defect also occupied

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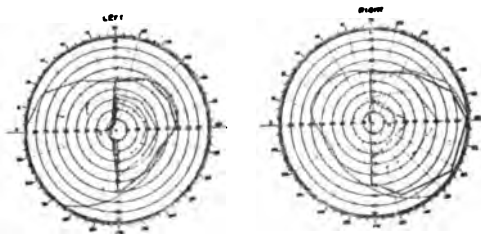
\* Reported in detail in *Medicine and Surgery*, Vol. ii, No. 5, May, 1918.

FIG. 11.



Case VI. Röntgenogram of head (one month previous to operation). Note large sella turcica; antero-posterior diameter, 25 mm.; depth, 19 mm. Note anterior wall projecting into sphenoidal sinus; posterior clinoid process almost obliterated.

FIG. 12.



Case VI. Perimeter charts showing bilateral homonymous hemianopsia.

FIG. 13.



Case X. Note general makeup of eunuchoid gigantism, abnormal development of long bones; absence of hair about torso and extremities, slightly atypical distribution of pubic hair; and absence of adiposity.

the temporal field. Later, both eyes became more or less involved at the same time, and this progressed with fluctuations. At times there was almost complete blindness for a short interval, at other periods vision in both eyes would be fairly good. Red splotches were seen on the white pages, before both eyes. During the last few months of the disease the previous attacks of diarrhoea have changed into marked constipation, with tendency to gaseous distention and abdominal pain. There has been transient defective hearing in the right ear. Slight fat padding in the mammary and axillary regions was noted, but no further increase in size of the hands occurred. One and one-half year previous to observation, or after one year of headaches, this symptom disappeared entirely for a number of months. When the headache stopped, there also was disappearance of fat padding. Eight months previous to observation, under Doctor Sihler's treatment, improvement of vision, decrease in size of the hands and feet, and in the gaseous distention, and general improvement in the entire condition were noted. Seven weeks previous to entrance into the hospital, the third increase of symptoms occurred. At that time the abdominal symptoms were greatly intensified, and the headache returned, but seemed different in character, described as severe, sharp pain in the midfrontal region above the bridge of the nose, accompanied by unrelievable pains in the arms and legs. With the onset of this exacerbation, the vision became much worse and progressive. When improvement in vision took place, it seemed, as described by the patient, as if a vertical curtain were slowly drawn from the nasal to the temporal sides of the eyes. At present the patient has an abnormally voracious appetite, without abnormal thirst. The *urine is normal in quantity* at the present time. Tendency to somnolence has returned with the last increase in symptoms. Two years ago, accompanying the second increase in severity of symptoms, the patient noticed a tendency to deviate to the right when walking with eyes closed, or when in an intensely bright light. This ataxia has continued to the present time. *Past:* Measles, mumps, whooping-cough, *typhoid-fever*, malaria. Five years previous to observation, the patient was "slugged" with a shot bag, the first blow being delivered on the left temporal, and another on the right parietal region, producing fracture (?) of the skull. There was



no period of unconsciousness following this trauma. *Personal and Family*: Unimportant.

*Examination.—General*: Very pale, anæmic man, 5 feet 11 inches ( $180\frac{1}{4}$  cm.) in height, 142 pounds in weight. Decidedly uncertain, ataxic gait. Musculature soft, atrophic, pseudo-œdematous; skin velvety, thick, elastic, dry; fingers and hands puffy, also arms and legs. Marked absorption of adipose tissue, skin seems loose and doughy. Slight puffiness about eyes. Very slight yellowish discoloration about face. No abnormal distribution of hair. Pulse, low volume and tension, tenotic type. Temperature (three days' observation),  $97^{\circ}$ – $98.2^{\circ}$  F. Pulse, 68–85. Respiration, 20–24. Blood-pressure, 110/80. Measurements: from head to symphysis, 34 inches ( $86\frac{1}{4}$  cm.); from symphysis to soles of feet, 37 inches (94 cm.); length of humerus,  $12\frac{1}{2}$  inches ( $31\frac{3}{4}$  cm.); forearm, 11 inches (28 cm.); femur, 18 inches ( $45\frac{3}{4}$  cm.); lower leg,  $17\frac{1}{2}$  inches ( $44\frac{1}{2}$  cm.). *Regional* signs practically negative. Testicles and penis normal size. *Urine* (twenty-four-hour specimens): 1300–2000 c.c., otherwise negative. (Note early history of polyuria.) *Blood* (including Wassermann) normal. *Carbohydrate tolerance* increased: Blood sugar after twelve hours' fast, 0.096 per cent. (normal, 0.10–0.13 per cent.); after 100 gm. dextrose and 1 c.c. of Adrenalin (1:1000) intramuscularly, with twenty-four-hour general diet, 0.08 per cent. *Spinal fluid*: Cell count 3, Wassermann negative. *Feces* negative. *Sella turcica* greatly enlarged (see illustration No. 11): antero-posterior diameter, 1 in. (2.5 cm.); vertical diameter,  $\frac{3}{4}$  in. (1.9 cm.). (Normal antero-posterior diameter is  $\frac{3}{8}$ – $\frac{1}{2}$  in. "1–1.2 cm.," normal vertical is  $\frac{3}{8}$  in. ".8–1 cm."). Posterior clinoid process almost entirely absent, so that there is large cavity merging with mastoid cells. Sphenoid sinus almost obliterated, posterior wall of sphenoid being pushed forward and downward. Large dark aræ above and posterior to sella turcica, measuring 2 in. in length and  $1\frac{1}{2}$  in. in height. *Perimeter examination* (observation of Dr. H. S. Hughes two years before present observation, soon after onset of visual signs): Bilateral homonymous hemianopsia. (See chart.) At time of this examination, right side of each visual field was almost completely blind. This blindness did not project into left visual field, but was limited by vertical line drawn through perimeter at its centre. There was no inversion of color fields in right eye.

Pupils were equal and reacted to light and accommodation. No disturbance of enervation of eyeballs or lids.

*Subsequent Course* (report of operation by Dr. Allen B. Kanavel, Chicago, April 26, 1917).—"I operated upon the patient and came down upon a mass in the neighborhood of the hypophysis which I suppose came from the hypophysis itself, but that is, of course, impossible to say. We scraped out a fair amount of the mass. A large amount of fluid escaped, so evidently it is more or less cystic, but there seemed to be proliferating tissue in addition to the cyst."

*Readmittance History* (May 7, 1918).—On April 26, 1917, the patient was operated upon by Dr. Allen B. Kanavel, at Wesley Memorial Hospital, Chicago. The patient showed signs of improvement, but was extremely weak for one or two months following operation. This weakness disappeared, and the symptoms of which he had previously complained were improved. For two months polyuria was absent and the condition in that respect was normal; then the patient again developed polyuria, which lasted for three months. During this time the urine was examined frequently, but at no time was there found any trace of sugar. After this, the polyuria disappeared, and the urine has been normal in amount. The slight swelling of the hands and feet also returned to normal. Previous to operation, the bones appeared to be enlarged, but now seem normal. The same sizes of shoe and glove are worn as previously, but they are much looser. Pains in the arms and legs have not entirely disappeared, although they are less severe. Before operation the patient had noticed subjective vibration of the eyeball (had never been told by anyone that this could be noticed). On account of this vibration, he would go about with eyes closed, as this sensation was absent when the lids were not open. This condition lasted for some time after operation, but has since disappeared, and has not recurred. Sight became improved after two months, and continued to improve for six months. The patient was able to distinguish objects, as the rung of a chair, or, in walking, the sidewalk; and if he knew a person, was able to recognize him; also could read the headlines of a newspaper. At present, however, he is able only to distinguish light from darkness with the right eye, and with the left eye is able to distinguish objects. At any time during the summer, following operation, he was able to tell the color of a house some distance away. At the present time the

headaches are less severe, involving the right side, beginning either posterior to the right mastoid process or in the right frontal region, and tend to respond more readily to treatment. Recently the headache has had greater tendency to originate in the right frontal region, very seldom occurring on the left side. The patient was free from these headaches until six months ago, when he contracted a cold, which remained practically during the entire winter; and while suffering from this cold, all symptoms tended to recur, but with less severity than previous to operation. Before operation the patient was a great meat eater and could not eat cakes, pies, etc., but at the present time has a desire for fruits rather than for meats. He has noticed during the past three months that the headaches have come on after eating. His appetite is ravenous, but he *has lost about 35 pounds in weight since operation*. In December, 1917, the patient returned to Chicago to consult Doctor Kanavel, who noticed pads of fat under both arms. He was then placed upon "Pituitary Gland," three times a day, by mouth, and under this treatment the fat pads disappeared. Ataxia has also disappeared.

*Reëxamination.*—*Physical signs* practically the same as on previous examination. *Blood sugar* (5-8-18), 0.076 per cent.; following 300 gm. dextrose given in twenty-four hours, by mouth, and two injections of Adrenalin of 8 minims, 0.08 per cent. *Röntgenogram of skull* (5-9-18) (transverse view): Antrum, frontal sinus, and nasal processes have been very slightly disturbed by operation. Sphenoidal sinus, anterior and posterior walls, easily defined. Entire sinus much clearer, less evidence of compression posteriorly than previous to operation, as shown in röntgenogram at that time. Posterior clinoid process entirely absent, so that there is almost straight line going from anterior wall of sella turcica back to mastoid cells above aural canal. No changes in other details of skull.

*Subsequent Course.*—Recent communications state that the patient is still living. There has been no improvement in sight, but some in general condition. There has been no additional development of adiposity or change in osseous growth; and no recurrence of polyuria.

CASE VII.—G. B., m., 18, Gen. No. 551. Referred by Dr. C. E. Matlock, Sligo, Mo.

*History.*—Two years ago fullness and weight in the head was

noticed, described by the patient as "feeling solid." Soon afterward *polydipsia*, *polyphagia*, and *polyuria* appeared. Intake of water was about two gallons daily. The urinary output was very large, but never definitely noted. The appetite was ravenous, the patient being able to eat a full meal and one hour later repeat the performance, being as hungry following a very heavy meal as before. Coincidentally he became subject to *headaches*, which were localized in a circle about 3 in. in diameter, in the mid-forehead, occasionally radiating through to the occiput, without definite localization about the bridge of the nose. This headache never became intolerable, and is described as dull pain, intensified by exertion, heat, and *lack of water*. Visual disturbance also occurred, described as "haziness," tendency to *bitemporal* type, markedly intensified by lack of water. Vision is improved by rotating the eyeballs downward, and is clear when directed straight downward. Visual disturbance is usually associated with headache. This is intermittent, vision at times being perfectly normal. There was some disturbance of pulse-rate, tachycardia, and cardiac palpitation, associated with vertigo and weakness, likewise intensified by lack of water and relieved by sufficiency of the same. Drinking of water relieves this entire complex for about two hours, when the patient must again have water to prevent recurrence of symptoms. There was also an increase in nervousness, principally in the form of irritability, tendency to worry, etc. The hands, arms, and feet would swell to twice normal size, the hands being so large that it was impossible to close the fist (from description, not œdema, no pitting being noted). This condition is more marked toward the close of the day. He seems to be much thinner and "pinched" in the early morning. Different observations have shown as much variation in weight as 10 pounds, between night and morning. The entire symptom-complex, consisting principally of polyphagia, polyuria, polydipsia, increased irritability, headaches, visual disturbance, vertigo, and cardiac palpitation, has progressively increased for about one year. The patient then began to be conscious of decided weakness, chiefly in the arms and legs, being unable to do an ordinary day's work or even to undergo slight exertion, such as walking for any length of time, without marked fatigue and shortness of breath. The polydipsia increased, the intake reaching 4 to 6 gallons of water a day, with polyuria in proportion, urination occurring every three hours, in varying quan-

tities, with nycturia once or twice. There was an increase in nervous symptoms, both mental and physical, tremor more marked, tachycardia more decided (bearing definite relation to supply or lack of water). The patient is emphatic in the assertion that all symptoms are increased by lack of water and that definite relief is produced by fluid intake. Six months ago he began to have decided dyspnoea. From the description, this does not seem to be a definite asthma, but the patient is forced to assume an upright position, a prone position causing aggravation of dyspnoea. Breathing is more rapid, and his brother states he believes the patient has been slightly cyanotic. This condition is also relieved by water, and apparently induced by lack of the same. Associated with this there has been no swelling of the neck, and no pressure symptoms retrosternally or other symptoms referable to the thymus. In the past three months, under treatment (exact nature unknown), there has been slight improvement in all symptoms. In the past six months there has been decided temperamental change, the patient having a tendency to become irritated by small noises, easily depressed, etc. *Past, personal and family*: Unimportant.

*Examination.—General*: Measurements: Vertex to symphysis, 32 in. ( $81\frac{1}{4}$  cm.); symphysis to soles of feet, 34 in. ( $86\frac{1}{4}$  cm.); span, 69 in. ( $175\frac{1}{4}$  cm.). Head rather small, fairly characteristic profile, slight recession of chin. Overcrowding of both upper and lower teeth, upper incisors slightly enlarged. Thyroid not enlarged, no padding in supraclavicular spaces, no mammary or girdle obesity. No abnormal pigmentation. No abnormal distribution of hair. Hair rather scanty in axillæ, fairly normal on mons. Eye-brows somewhat thinned at outer thirds. Hair on face and head normal. Hands fairly characteristic, fingers very short and stubby, wrists slightly wide. Long bones fairly well developed. Pulse, slow and normal. Temperament that of depression. Temperature,  $99^{\circ}$  F. Blood-pressure, 135/95. *Urine* (twenty-four-hour specimens): 1000–10,000 c.c., sp. gr. 1008–1012, faint trace of albumin, otherwise negative. *Blood* (including Wassermann) normal, except slight secondary anæmia. Blood sugar, 0.096 per cent. (normal, 0.10–0.13 per cent). *Sella turcica* normal.

*Reaction to Treatment.*—The mental condition was greatly improved, the polyuria not affected by tolerant doses of Pituitrin.

CASE VIII.—M. H., f., thirty-two, Gen. No. 1357. Referred by Dr. Wm. S. Chase, Akron, O.

*History.*—Present symptoms began ten years ago, gradually, with frontal *headaches* and *polyuria*. The headaches were the first ever experienced, and urinary disturbance had not been previously noted. The exact quantity of urine at onset is not recalled. Weight was 160 pounds, reduced in two months to 140 pounds. It then increased, reaching 150 pounds in four months. Typhoid fever occurred at the age of fourteen (in bed thirteen weeks), following which the patient gained weight rapidly, weighing 160 pounds between the ages of fifteen and sixteen, which weight persisted for six years. Shortly after onset of polyuria a definite polydipsia was noted. Six or eight years ago, urticaria began and has since occurred practically every evening. For the past ten years the patient has had gastric disturbance, nausea and vomiting, particularly during menses. Thirteen years ago an exploratory operation for "appendicitis" was performed, but the appendix was not removed. Four years ago a second operation for abdominal pain was performed, the appendix and right ovary removed. Pain and tenderness have not returned, although the patient is still troubled with eructations. Eight years ago auditory disturbance began in left ear, gradually increasing until hearing on the left is absent and there is beginning involvement of the right. Five months ago polyphagia began. Nausea, vomiting, and hematemesis occur, relieved by eating. She eats as often as every hour in order to relieve nausea. There is considerable pyrosis. Polyuria has been intermittently present for the last ten years, more marked at times than at others. The amount of urine passed is not known definitely, but more than normal. The menses have been absent for the past four months, but epistaxis has occurred at menstrual time. The periods began at eighteen, scanty, three days' duration, always painful, accompanied by nausea, vomiting, and fainting attacks, demanding rest in bed. The patient was normal at birth. The teeth began to erupt at three months; the ages of walking and talking not recalled. She was always well and healthy until fourteen, when she had typhoid fever. Temperature is always abnormal, 97.6° F. Occasional cardiac palpitation and dyspnoea on exertion occur. Headaches are present almost constantly, frontal and occipital in character. There is some vertigo. The patient hears better in a noisy room than in a quiet one. The skin bruises easily, is

dry and somewhat rough and scaly, easily irritated, itchy, and sensitive. The dentine is hard, finger-nails hard and brittle. The thyroid gland at times becomes enlarged, usually during period time, which causes no great concern. The hair has not fallen out, or changed in pigment. Vague pains occur in the back, between the shoulders, and in the upper right quadrant of the abdomen, never colicky or associated with jaundice, but frequently with distention, gurgling, and belching. *Past*: Measles, scarlet fever, mumps, whooping cough, chicken pox. Tonsillitis once, twelve years ago. Typhoid fever at the age of fourteen. Operations as described above. *Personal*: Appetite good. Bowels regular. Patient sleeps fairly well. Three cups of coffee a day. *Family*: Father dead, aged fifty-five, pneumonia. Mother died at the age of thirty-eight, "stomach trouble." Father and paternal grandmother were both deaf. Mother's sister had diabetes.

*Examination.—General*: Upper measurement,  $29\frac{3}{4}$  in. ( $75\frac{1}{2}$  cm.); lower,  $31\frac{1}{2}$  in. (80 cm.); span, 66 in. ( $167\frac{1}{2}$  cm.). Eunuchoid type; head, hands and feet not characteristic. No unusual amount of adiposity, mammae not large. Hair distribution well localized. Slight chloasma about eyelids and forehead, no other marked pigmentation. Pulse normal. Blood-pressure 104/85. Erythema produced by slight irritation, about arms, back, and limbs. Temperament psychoneurotic, well controlled. Secondary sex characteristics normal. *Regional signs* normal. *Urine* (twenty-four-hour specimen), 4080 c.c., sp. gr. 1010, acid, light lemon color; trace of albumin, sugar negative, microscopic negative, except many small epithelial cells. Single specimens, sp. gr. varies from 1003 to 1010. *Blood* (including Wassermann) normal. *Stomach contents* (Ewald breakfast), 50 c.c., tinged with bile; free HCl 40, combined acidity 9, total acidity 49; microscopic, yeast cells. *Carbohydrate tolerance* increased: Blood sugar after fifteen hours' fast, 0.10 per cent. (normal, 0.10–0.13 per cent.); one hour after 101.5 gm. dextrose, 0.082 per cent. (normal, 0.18 per cent.); two hours after same, 0.116 per cent. (normal, 0.15 per cent.). *Basal metabolism*, +12 per cent. *Fluoroscopic of stomach* negative. *X-ray of sella turcica* normal. *Fluoroscopic of colon*: Mild spasticity of descending portion.

*Results of treatment* unknown.

CASE IX.—H. B. S., m., twenty-six, Gen. No. 2327.

*History.*—Polyuria was first noticed one year previous to observation, reaching a maximum of one gallon (4000 c.c.) in twenty-four hours, without nycturia. Polydipsia was present, without polyphagia. The next symptom noted was a peculiar *paresthesia in the mid-forehead*, about an inch above the bridge of the nose, described as a “tickling sensation, running up and down” in this region, lasting three or four weeks. He then began to note a slight increase in nervousness, a feeling of *high tension*, of being *on edge*. He was inclined to “go to pieces” rapidly, particularly during the discussion of such topics as death. There was tendency to *increased irritability* and worry, but no emotional disturbance. There were no physical nervous manifestations. He was conscious of *headaches in the mid-frontal region* after a hard day's work at books, of same intensity as it had been for the previous four or five years. The appreciable *failure in visual acuity* became more marked, requiring glasses. Near vision became more indistinct, not, however, affecting any particular portion of the visual fields. These symptoms have been gradually increasing in severity, particularly for the past month. The patient *at no time has noticed any perceptible change in the hands and feet*, or wider separation of the teeth, the upper central incisors having always been separated. He sleeps well, and has no tendency to diurnal drowsiness. No signs or symptoms referable to other systems. *Past*: Measles, severe tonsillitis (followed by rheumatism). Since tonsillitis and rheumatism, the patient has been complaining more or less of the symptoms above. *Personal and family*: Unimportant.

*Examination.—General*: Slightly disproportioned young man, overdeveloped, particularly the arms and legs. Height, 5 ft. 7 in. (170 cm.); weight, 160 pounds. Musculature well developed throughout, of good tone. No abnormal localized deposition of fat. Mammæ rather prominent. No abnormal distribution of hair on the chest or abdomen; very fine, scanty hair on the extremities. Very slight chloasmic pigmentation about the forehead, extending down over the face to the collar line, not corresponding to the exposed portions. Number of brownish verrucæ scattered over the body. Temperament, psychoneurotic, well controlled. Blood-pressure 140/70. Temperature, 97.8°–99° F. Pulse, normal, 76–90 (three days' observation). *Positive regional signs*: Marked separation of the upper



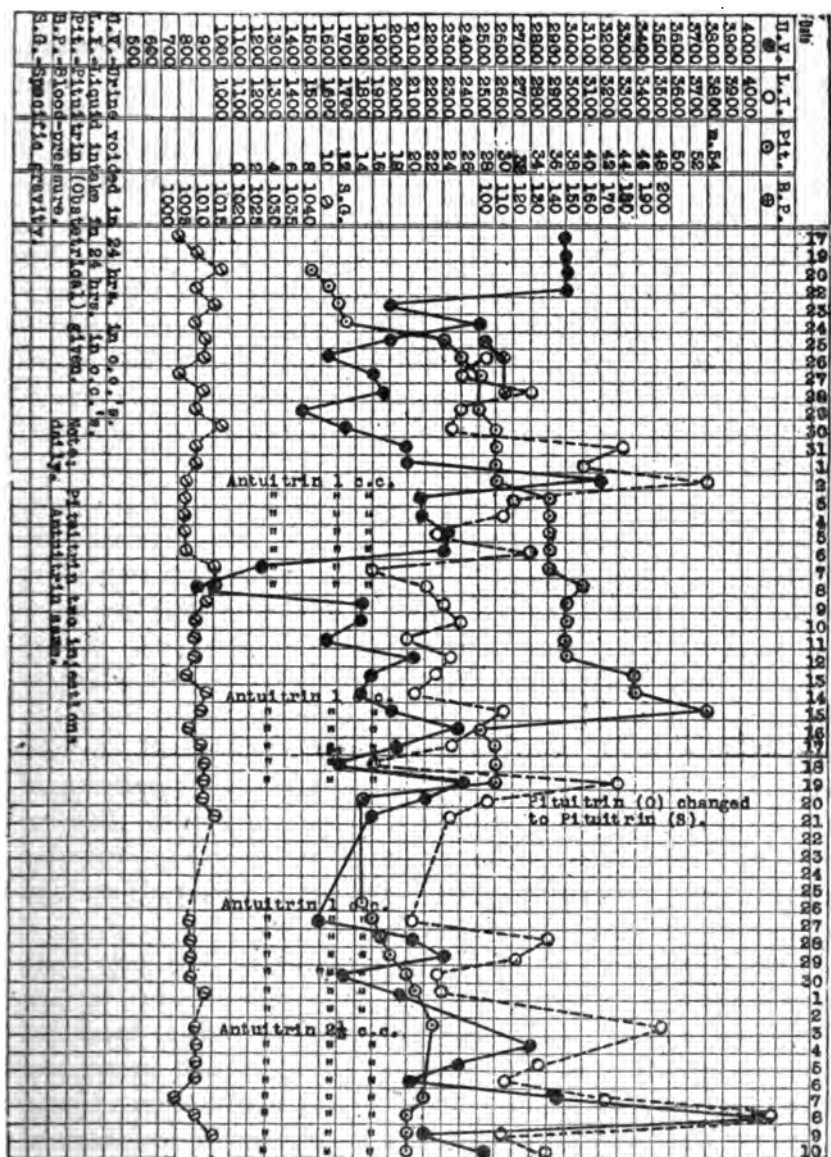
teeth. *Heart*: Apex beat in the fifth interspace, within the nipple line. No thrill, or rub palpable over the precordium or sternum. No diastolic shock. Apex beat, third rib, midsternal line. Sounds, apex—*distinct systolic murmur* accentuation of the second tone. This murmur is transmitted beyond the apex, about midway to the anterior axillary line, and almost completely disappears with deep inspiration. Same murmur heard over the pulmonic area, disappearing entirely on deep inspiration. Disappearing murmur also heard over aortic area. Second pulmonic markedly accentuated; second tone over aortic area also accentuated. No abnormal findings about large vessels of neck and chest. *Urine* (twenty-four-hour specimen): 2975–3125 c.c., pale, sp. gr. 1001–1010, otherwise negative. *Functional kidney test* (phthalein): two hours, 50 per cent. *Feces* normal. *Blood* (including Wassermann) normal. *Fluoroscopic of chest and stomach* negative. *Colon* spastic. *X-ray* of sella turcica slightly large, but not abnormal.

*Result of Treatment*.—Not obtainable.

CASE X.—C. H. D., m., twenty-five, Gen. No. 1195, referred by Dr. B. S. Rose, Evansville, Ind.

*History*.—Symptoms of increased fatiguability came on gradually two years previous to observation without special cause. This condition increased, until he was unable to do any work. Other symptoms were mental retardation and aching over the entire body, *head-aches*, constant, *bitemporal* and *midfrontal*. During the last year the *urinary output* has been *increased*, found, when measured, to be one pint an hour (during the day) with two to six *nycturia*. Definite *polydipsia* and *polyphagia* (especially for sweets) was present. Sleep is not restful. He has lost twenty pounds in weight during the last one and one-half years, always feels chilly and has frequent cardiac palpitation. Intercourse definitely aggravates all symptoms. Frequent discomfort over bladder due to spasm, also tenderness in epigastrium, with belching. Bowels usually move daily, sometimes several times a day, but occasionally are irregular. First tooth erupted between the first and second months. Patient states he has grown rapidly, increase in height most marked at the age of fourteen or fifteen. He has not grown during the last four or five years. *Past*: Diphtheria, measles, mumps, pneumonia twice. Injury nine years ago to the frontal region of the head, leaving a slight abrasion. Phy-

FIG. 14. CASE X.



sician at that time stated there was no fracture of the skull. Depression is visible in this region, and also slight one over parietal region. *Personal*: Appetite good. Patient sleeps fairly well. Three cups of coffee a day, no alcohol. Ten cigarettes a day. Neisser infection five years ago. *Family*: Father well, mother nervous.

*Examination.—General*: Upper measurement,  $35\frac{1}{4}$  in. ( $89\frac{1}{2}$  cm.); lower,  $38\frac{3}{4}$  in. ( $98\frac{1}{4}$  cm.); span, 77 in. ( $195\frac{1}{4}$  cm.). Eunuchoid giant makeup, large hands, face more of eunuchoid type, although hair distribution is good. No marked hair distribution about body. Long bones markedly overdeveloped in length. No adiposity. Pulse normal. Blood-pressure, 90/60. Temperament psychoneurotic, fairly well controlled. *Cystoscopic and ureteral catheterization* (by Dr. Leo Bartels): Bladder, prostate, and pyelogram of pelvis of kidneys negative. *Urine* (twenty-four-hour specimens): 3000–5000 c.c., sp. gr. 1005–1015, color light, trace albumin, dextrose and microscopic negative. *Blood* (including Wassermann) normal. *Functional kidney test*: Total (two hours), 45 per cent. *Carbohydrate tolerance* increased: Blood sugar after fifteen hours' fast, 0.117 per cent. (normal, 0.10–0.12 per cent.); one hour after 122 gm. dextrose, 0.195 per cent. (normal 0.16 per cent.); two hours after same, 0.162 per cent. (normal, 0.10–0.13 per cent.). *Spinal fluid*: Cell count 3, globulin positive, Wassermann negative. *Gastric contents* (Ewald breakfast): Free HCl 2, combined acidity 0, total 2, microscopic negative. *X-ray of skull*: Sella turcica small, anterior and posterior clinoid processes almost in contact. *Fluoroscopic of stomach*: Dilated stomach, aerophagia; duodenum normal. *Electrocardiograms* negative except for mild suggestion of right ventricular preponderance. *Basal metabolism*, 3 per cent. *Pituitrin intestinal reaction* obtained with 20 minims of Pituitrin (0).

*Results of Treatment.*—Polyuria, polyphagia, and polydipsia were decreased to practically normal, the amount of urine varying from 1500–2500 c.c. Other general symptoms, such as headache, extreme fatiguability, bladder spasm and irritability and spasms, and loss of weight, were not affected by Pituitrin treatment. Symptomatology was practically unchanged by Adrenalin treatment or Suprarenal Substance gr. 5–10 four times a day, and Adrenalin (1:1000) minims 30–40 given intravenously did not either increase or decrease polyuria. Institution of Anterior Lobe Pituitary Gland

or Pituitary Substance (containing both anterior and posterior lobe extracts) by mouth, with a combination of anterior and posterior lobe extracts hypodermically, had no more decided effect upon the general condition, although the polyuria was reduced to the same amount as that obtained from posterior lobe substance alone. Combination of anterior and posterior lobe pituitary substances, Adrenalin and Orchitic Substance, had no more effect than the other treatment mentioned above. When the patient was taken off posterior lobe pituitary substance, the polyuria was increased from 3200 to 5000 c.c. Extract of Placenta gr. 10 four times a day made no impression upon the polyuria, or general condition. Twelve X-ray exposures of the hypophysis produced no change in polyuria or other symptoms.

CASE XI.—H. F., m., fifty-two, Gen. No. 1614.

*History.*—The history in this case dates over a period of forty years, so only a brief summary can be given. Onset occurred at the age of twelve, with marked frequency of urination, micturition occurring as frequently as every half hour, with considerable nycturia. At the age of fourteen a diagnosis of diabetes insipidus, with "neurasthenia torpida" was made by Dr. Louis Braemer. From that time until the age of twenty-two this polyuria was associated with polyphagia, polydipsia, and marked hypersexuality. At the age of twenty-two he suffered from an attack of nervous exhaustion (?), incapacitating him for more than a year. A sojourn in California for this condition relieved him of the *muscular pains* in the thighs (which was his most distressing symptom) in two months, and the polyuria in four months. Retinal hemorrhages occurred at the age of twenty-one, and four attacks of this condition have occurred during the following twenty years. Many eye symptoms, some of which were conjunctivitis, keratitis, "migraine ophthalmic," errors of refraction, and astigmatism, have been constant complaints throughout the entire course. High altitudes produced considerable relief of all ocular symptoms. The polyuria has persisted throughout the entire course of forty years, but it was absolutely unaccompanied by glycosuria until the age of thirty-seven. Previous to that time an artificial glycosuria had been unsatisfactorily attempted, by giving the patient large amounts of glucose. At the age of thirty-seven, glycosuria was first discovered during an examination for life insurance. This glycosuria has persisted more or less constantly from the age of thirty-

seven to fifty-two, as high as 8 per cent. being present during a stage of excessive polyuria. At the age of forty-five, the first test for hyperglycemia was made, which ranged from 0.18 to 0.20 per cent. The hyperglycemia was entirely out of proportion to the glycosuria. For instance, after the patient had been on a restricted diet, which would clear the urine entirely of dextrose, the blood sugar would vary from 0.129 to 0.20 per cent. Diacetic acid and acetone were present only on one occasion. During the last few years the glycosuria has been much more difficult to control with dietetic measures, and the blood sugar has gradually increased to from 0.20 to above 0.30. The polyuria has fluctuated decidedly during this time, but very rarely has been below 7000-8000 c.c. The maximum amounts have been 10,000-20,000 c.c. of urine daily. As early as fifteen or sixteen, the patient showed acromegalic changes in form of an unusually long torso and short, muscular legs and arms. At that time he was unusually athletic, and prided himself on his acrobatic feats of strength. Growth was practically arrested after the sixteenth or seventeenth year, and no special change occurred in the acral, or short and flat bones. Slight stiffness and fullness of the terminal phalanges have been present at frequent and variable intervals. No marked adiposity occurred until the last few years, when there has been marked tendency to gain in weight. Muscular tone and strength, formerly excessive, have gradually decreased in the last few years, so that now the patient has an exceptionally distressing fatigue, frequently being so tired and exhausted, both mentally and physically, that he is required to go to bed. Hypersexuality has been continuous from the first, and no decided loss of libido has been present, although at times the muscular fatigue is so great that it interferes with sexual desires. No conception has occurred during a married life of over thirty years. Nervous instability and lack of concentration have been constant since boyhood. An occasional mild brainstorm, in which there is marked loss of concentration, memory and orientation, with considerable dissociation of ideas and mental confusion, has occurred. A number of typical attacks of gall-bladder trouble, sinusitis, septic sore-throat, and what was thought to be renal colic, have occurred since the age of thirty. He has a marked susceptibility to cold, as noticed particularly in the early course of the disease. *Past:* Eczema of the scalp since birth. Scrofula in early childhood. Acute articular rheumatism

FIG. 15.



Case XI. Note increased length of torso as compared with extremities: from symphysis to vertex, 86 cm.; symphysis to soles, 79 cm. Note acromegalic character of hands and feet. Slight adiposity of girdle type, with hypogastric and mons padding.

FIG. 16.



Case XI. Note acromegalic character, broadening of wrist and palm, and short, somewhat blunted fingers (tendency to spade-hand).



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at the age of thirteen. Varicose veins in early adult life. *Personal*: Moderate use of narcotics. *Family*: Father acromegalic. Mother eunuchoid, suffered from migraine, and died of early arteriosclerosis. One brother very obese. One sister acromegalic, having migraine, refractive ocular symptoms, and nervousness. One sister very stout, with short stature, extreme psychoses.

*Examination.—General*: Typical acromegalic stature; short, stocky, with marked overdevelopment of musculature. Head large, classical in type, with pituitary nose, pointed chin, and very prominent superior maxillary process. Hands short, stocky, "type en large," slight clubbing of finger-tips, white crescents barely visible. Feet of same character. Marked abnormal distribution of hair across abdomen. Eye-brows not bushy, temporal halves thin. Hairline at temporal region  $2\frac{1}{2}$  cm. from eye-brow; frontal region, 6 cm. Hair on face not unusually thick, mustache medium. Unusual hair distribution on lower third of upper arm; no abnormal amount on lower extremities. Hair of axillæ and mons normal. No abnormal pigmentation. Tendency to diffuse erythema about head, no erythematous spots on rest of body. Few small angiomas scattered over abdomen and chest. Pulse normal. Measurements: Height, 65 in. (165 cm.); from head to symphysis, 34 in. (86 cm.); from symphysis to soles of feet, 31 in. ( $78\frac{1}{2}$  cm.); span, 66 in. (168 cm.); bridge of nose,  $5\frac{1}{2}$  cm.; length of humerus, 11 in. (28 cm.); forearm,  $9\frac{1}{2}$  in. (24 cm.); femur, 13 in. (33 cm.); lower leg,  $13\frac{1}{2}$  in. (34 cm.); circumference of head, 22 in. (56 cm.); at upper maxilla, 21 in. (53 cm.); at lower maxilla, 21 in. (53 cm.); neck,  $17\frac{1}{2}$  in. (44 cm.); chest,  $39\frac{1}{2}$  in. (100 cm.); abdomen, at navel, 34 in. (86 cm.); symphysis, 36 in. (91 cm.); upper third of thigh,  $21\frac{1}{2}$  in. (55 cm.); calf, 14 in. ( $35\frac{1}{2}$  cm.). In lying position no girdle or mons obesity, no distinct fullness of mammæ. No supraclavicular fat pads, no cushions about backs of hands or feet. Mentality, psychoneurotic, well controlled. Temperature,  $98.8^{\circ}$  F. Blood-pressure, 150/100. *Regional*: None except osseous changes referable to acromegaly. *Urine* (twenty-four-hour specimens) varied from 2400 to 20,000 c.c.; sp. gr. (daily variation of two-hour specimens) from 1000 to 1030, the highest occurring at 5 A.M.; average sp. gr. of twenty-four-hour specimens from 1010 to 1012; albumin negative, dextrose present in variable amounts (as stated above); occasional granular cast, other-



wise negative. *Sugar tolerance* decreased: Blood sugar following twelve hours' fast, 0.183 per cent. (normal, 0.10–0.13 per cent.); two hours after 142 gm. dextrose, 0.364 per cent. In recent years, blood sugar after twelve hours' fast, 0.27–0.30 per cent. *Basal metabolism*, +6.21 per cent. *Blood* (including Wassermann) normal. *Electrocardiogram*: Relatively small deflections in all three Leads; "T" inverted in Lead 3, split "QRS" complex in Lead 3. *Sella turcica* normal size compared with skull; 10 by 16 mm. *X-ray of hand*: Metacarpal and phalangeal bones very thick, dense, and short; decided inushrooming of ends of terminal phalanges, exostosis on terminal thumb phalanx; wrist wide, hand very short and pudgy.

*Diagnosis*.—Bilobar pituitarism, preadolescent, non-neoplastic type, heteroactivity. Originally this was a case of hyperactivity of both lobes of the pituitary gland, considering the pars intermedia as a part of the posterior lobe. Recently there is evidence of a hypoactivity of the anterior lobe, existing with a hyperactivity of the posterior lobe, which would place this case into the heteroactive group of bilobar pituitarism.

*Results of Treatment*.—Very little treatment directed toward the pituitary disorder has been given in this case. Dietetic treatment directed toward the glycosuria and also the polyuria has been influential in controlling the glycosuria and decreasing to a slight extent the polyuria. The hyperglycemia has never been decidedly affected by dietetic treatment consisting of periods of starvation of four to six days' duration. On one occasion the patient received a dose of Adrenalin with a local anæsthetic, and noted an enormous increase in the polyuria for a number of days, which commenced within a few hours after the injection of Adrenalin. This was accompanied by a very unusual hypersexuality, during which there was constant priapism for a few hours. Cocaine, which had been applied in 20 per cent. solution, locally to the mucous membranes and injected into the gums, had not had any effect upon the signs or symptoms. A few small doses of Pituitrin (0) (minims 5, Parke, Davis & Co.) have been given, and were constantly followed by a marked increase in polyuria, so this treatment was not thought advisable. Three doses of Antuitrin (Parke, Davis & Co.) of 1 c.c. each apparently produced considerable effect upon the muscle and mental fatigue, and following each injection there was slight increase in the polyuria. A high

altitude (5000 to 8000 feet above sea level) on several occasions produced a very marked amelioration of all symptoms. The local symptoms of the eyes, the marked nervous disturbances, and the polyuria, polyphagia, and polydipsia, were all very favorably affected by a high altitude.

#### DISCUSSION OF REPORTED CASES

An analysis of the above 11 cases with regard to incidence of the disease, demonstrates the already known fact that this syndrome occurs more frequently in the male, 10 of the 11 cases being of this sex, and that it is more prevalent in early life, all of the cases of this series having begun before the age of twenty-eight. Those cases having their onset after this age are more likely to be associated with hypophyseal neoplasm. Heredity could not be held accountable in the series as a predisposing factor. In only a few of the cases, for example, those having pituitary tumor (Cases V and VI) and those having gigantism and acromegaly (Cases III and XI), the family history played an important rôle. Infectious disease and cranial trauma occurring in early adult life are probably etiological factors as demonstrated in the histories of Cases I, IV, V, VI, and X.

In this paper it is deemed too academic to discuss in a differential way intermittent and chronic polyurias of other types, such as those due to excessive drinking, absorption of transudates and exudates, hydronephrosis, bladder obstruction, contracted kidney, reflex hydruria following ureteral catheterization, *urina spastica* of migraine, epilepsy, etc. It is very pertinent, however, to discuss the etiological diagnosis and the pathogenic relationship of polyuria to the hypophysis. If such association can be shown a more penetrating consideration is the determination of the relationship of polyuria to the function or secretion of the *individual* lobes of this interesting gland. And should the lobar pituitary theory as an etiological basis prove tenable, then further studies will become necessary to demonstrate the *activity*, hyposecretory or hypersecretory, of the lobe of the hypophysis producing the distressing syndrome.

On the basis that this *polyuria and associated symptomatology are due to pituitary disorder*, it is most interesting to analyze the positive signs or symptoms *other than the increased diuresis and nervous symptomatology* present in these cases, that are definitely related to a

## SUMMARY OF CASES.

No.	Initials	Gen. No.	Age	Sex	Activity of lobes of Hypophysis	B. M. R.	Carbohydrate tolerance Before 1st hour 2nd hour	Blood- pressure Syst. Dias.	Treatment	Response and comment
I	F. B.	1203	28	M	Eunuchoid giant, pre- adolescent anterior lobe hyper-	- 3	.130 .156 .112 (normal)	120 85	Pituitary liquid "H"	Polyuria decreased. Sp. gr. increased; nausea, vomiting, and muscle fatigue relieved. Polyuria reduced; sp. gr. increased; weakness and depression im- proved. In polyuria; decrease in libido; no improvement in head- ache or muscle fatigue.
II	C. K.	911	23	M	No signs or symptoms of pituitarium ex- cept polyuria	.....	.090 .096 .093 (increased)	110 75	Pituitary liquid "H"	
III	A. H. S.	1115	31	M	Acromegalic giant; preadolescent an- terior lobe hyper-; postadolescent an- terior lobe hyper- (transition to hypo-)	- 1	.087 .131 .108 (normal)	119 90	Antuitrin and pituitrin "H"; pituitary sub- stance, entire gland (oral)	
IV	C. W. F.	1194	32	M	No signs or symptoms of pituitarium ex- cept polyuria	- 8	.105 .144 .096 (normal)	118 92	Pituitrin "H"	Polyuria reduced (re- lapse on cessation of treatment). Weakness and headache relieved. Death (neoplastic).
V	J. N.	948	14	M	Pituitary neoplasm	.....	100 gm. dextrose did not produce glycosuria (.096 (after fast) (After 100 gm. dextrose by mouth and 1 c.c.; ad- renalin (1 : 1000) "H") .080 (increased)	.....	None	Improvement
VI	H. B. T.	1622	35	M	Pituitary neoplasm	.....	.096 (normal)	110 80	Operation	
VII	G. B.	551	18	M	No signs or symptoms of pituitarium ex- cept polyuria	.....	.10 .082 .116 (increased)	135 95	Pituitrin "H"	Mental state improved polyuria not influ- enced.
VIII	M. H.	1357	32	F	No signs or symptoms of pituitarium ex- cept polyuria	+12	.....	104 85	None	Unknown.
IX	H. B. S.	2327	26	M	No signs or symptoms of pituitarium ex- cept polyuria	.....	.....	140 70	Not known	Unknown.
X	C. H. D.	1195	25	M	Eunuchoid giant; pre- adolescent anterior lobe hyper-	- 3	.117 .195 .162 (normal)	90 60	Antuitrin and pituitrin "H". Oral substitu- tion of following: pi- tuitary substance, en- tire gland; suprarenal substance; orchitic substance; extract pla- centa. Adrenalin "H". x-ray treatments; spi- nal puncture	Polyuria, polydipsia and polyphagia decreased by pituitrin. Other symptoms not relieved
XI	H. F.	1614	53	M	Acromegaly, glycosu- ria, hyperglycemia hetero-lobar activ- ity; postadolescent anterior lobe hyper- (transposed to hypo-); postadoles- cent posterior and intermediate lobar hyper-	+6.21	.183 (decreased)	150 100	Very little pituitary medication. Dietetic controls glycosuria, not glycaemia	Adrenalin "H" produced enormous polyuria and hypersexuality. Co- caine negative; pitui- trin (0) m. v. produced increase in polyuria and glycosuria. Antui- trin produced im- provement in mental and muscle fatigue, no effect upon polyuria.

pituitary dyscrasia. Of the above cases, 5 had no definite osseous, adipose, or general changes, or other hormonal signs that would relate them specifically to pituitary disorder. On the other hand, 6 had very striking pituitary signs other than the polyuria and nervous manifestations. Two (Cases V and VI) were pituitary tumors, in which the polyuria was the initial and early sign. Case III had other signs of pituitary disorder, evidenced by the easily discernible osseous changes of *acromegalic gigantism*, having all the classical symptoms of acromegalia and gigantism associated with both pre- and post-adolescent hypersecretion of the anterior lobe of the pituitary gland. Cases I and X had, besides the pituitary polyuria complex, the classical hormonal signs produced by hypersecretion, during pre-adolescence, of the anterior lobe of the pituitary, responsible for the eunuchoid gigantism. In both these cases, other signs of disorder of the pars nervosa, such as glycosuria, hyperglycemia, and adiposity, were absent. In Case XI there were classical markings of acromegaly present with the onset and throughout the course of the polyuria (from the age of twelve to thirty-seven). In this case, *after a polyuria of twenty-five years' duration*, there occurred a glycosuria, hyperglycemia, and decreased sugar tolerance. This case then presented evidence of associated anterior lobe hyperfunction (acromegaly) during its initial course, and a related hyperfunction (glycosuria and hyperglycemia) of the pars nervosa during the late course of the polyuria. None of our cases showed adiposity, although the association of dystrophia adiposogenitalis (Froelich's syndrome) with polyuria has been reported by Biedl,<sup>37</sup> Fearnside,<sup>61</sup> Marañon and Pintos,<sup>62</sup> and Schünemann.<sup>63</sup> Theoretically, on the assumption that dystrophia adiposogenitalis is due to an insufficiency of both the anterior and posterior lobes, and that polyuria is due to a hyposecretion of the pars intermedia, the association should be more common.

An estimation of the basal metabolism in this series furnished comparatively little diagnostic evidence relating this syndrome to the pituitary gland or markedly disturbed activity of any lobe of this gland. The basal metabolic rate was practically within the normal in this series, varying from -8 per cent. to +12 per cent. The carbohydrate tolerance was normal in 6, increased in 3, and decreased in 1. The case in which it was decreased, an exception to the rule, was one of hyposecretion of the pars intermedia, thought to be the

cause of this syndrome, in which acromegaly, glycosuria, and hyperglycæmia were associated with a previously existing polyuria (Case XI). The blood-pressure tended to the subnormal. In 4 cases it varied from 90 to 110, in 2 cases it was slightly above the normal for the age, and in the other 3 cases it was within the normal variation.

The results of treatment, particularly of Pituitrin, demonstrated that in 5 cases this preparation exerted a decided influence upon the polyuria and other symptomatology. In 4 cases, including the 2 neoplasms, Pituitrin was not instituted. In 1 case this treatment had absolutely no effect upon the polyuria or other symptomatology (Case X). In this case there was an associated disorder of the anterior lobe, producing eunuchoid gigantism, which might account for the lack of response to Pituitrin treatment. A similar case (I), however, did react completely to this treatment. In 1 case (XI) it had an opposite effect, not ordinarily expected; namely, the production of an increase in the polyuria and associated symptoms. In this case there was clinical evidence of hypersecretion of the posterior lobe, associated with glycosuria, hyperglycæmia, decreased sugar tolerance, and acromegaly. In 1 case (XI) Adrenalin seemed to greatly intensify the polyuria, and in another (X) it was impossible to note any effect from Adrenalin or Suprarenal Substance, either upon the polyuria or associated symptomatology. In this same case (X) Extract of Placenta, a combination of Anterior and Posterior Lobe Extracts, Orchitic Substance, Thyroid Gland, and combinations of these different ductless-gland preparations, had no effect upon any of the symptoms. Cocaine solution was tried upon 2 cases, with practically no influence in activating or decreasing the symptomatology. Spinal puncture<sup>64, 65</sup> and X-ray radiations (ten exposures) of the hypophysis, applied to the refractory case above, not reacting to any ductless gland therapy, had absolutely no effect in ameliorating any of the symptoms.

Deductions from an analysis of this series of 11 cases show that in practically 50 per cent. there was evidence of decreased function or activity of the pituitary gland, affecting probably the pars intermedia. These conclusions are deduced (1) from the fact that not all cases have other evidence of anterior or posterior lobe disorder, as shown by the 5 cases described above, and (2) from the observation that only 50 per cent. of the cases were improved by Pituitrin treat-

ment. Why one classical case was not at all affected by Pituitrin treatment is difficult to explain, except on the ground that it is a bilobar case, having anterior lobar, in addition to pars intermedia involvement. The fact that the polyuria associated with the acromegaly (Case XI) was not relieved by Pituitrin treatment might also have been due to the specific activity of other lobes of the gland, for in this case there was decided involvement of both the anterior lobe and pars nervosa, as shown by the classical signs present resulting from disturbed function of these other portions of the gland. These two cases, however, help to contend against the theory that all cases are due to hyposecretion or decreased function of the posterior lobe or the pars intermedia of this gland, and demonstrate that there is a considerable amount of uncertainty regarding the activity of the hypophyseal lobe producing this interesting syndrome.

The many cases reported in the literature which have reacted to Pituitrin (the extract of the posterior and middle, or infundibular, lobes) of the *hypophysis*, have established a new era in the treatment, as well as in the etiology and prognosis of this disease. While it is true that nearly all investigators have found this Pituitrin treatment beneficial in the majority of cases, yet it is the opinion of the writers that it is not a specific, or even effective in all cases. In those cases in which it does produce marvellous changes in the symptomatology and course of the disease, the majority of investigators are free to admit that, unless it is continued indefinitely, there is tendency to relapse, and an interruption of treatment for any length of time frequently submits the patient to a return of all the symptoms of the disease. Only in Case I of the above series, after the syndrome had been completely relieved by subcutaneous treatment, was the patient able to continue on Pituitary treatment by mouth without a return of the symptoms. In this case, after he discontinued the oral administration of Pituitary Substance, there was a return of both the polyuria and nervous manifestations, although in a much milder form than those present previous to the initial treatment. The other 5 cases which reacted satisfactorily to intensive Pituitary treatment, had a constant tendency to relapse as soon as there was a reduction of the tolerant dosage of this substance. The fact that all these cases do not react to this treatment, however, and that even those that do react may relapse if the treatment is discontinued, should not prevent the

application of this form of therapy. The definite determination of the pathogenesis of this disease and its more favorable therapeutic response will depend to a large extent upon such clinical investigation as the very careful observation of the therapeutic reaction to Pituitrin and possibly other ductless-gland preparations in those cases of pituitarism in polyglandular syndromes.

Another very important estimation to be accurately noted in the administration of Pituitrin to these cases, which will undoubtedly help to clarify both etiology and treatment, is the careful measurement of the amount of Pituitrin each individual will tolerate without the production of its physiological effect. The writers have recognized and reported three types of physiological reaction, the "intestinal," the "vascular," and the "general." The "intestinal" reaction to Pituitrin evidences itself by a cramping, colicky sensation in the abdomen, succeeded within a few minutes by bowel evacuation. This "intestinal" reaction usually begins within five minutes and rarely occurs later than fifteen minutes after the injection of a sufficient amount of Pituitrin. The average individual having a normal secretion of the posterior lobe and pars intermedia, will obtain this reaction from 10 to 15 minims (1 c.c.) of Pituitrin (0). Doses which do not produce this reaction are considered insufficient to be effective in substituting for the normal amount necessary to the individuals possessing deficient hypophyseal secretion. It has been thought that the size of the dose producing this reaction might be used as a diagnostic means in determining the degree of insufficiency of the pars intermedia and posterior lobe of this gland. In other words, those individuals having a marked decrease in secretion, productive of the symptomatology of pituitary polyuria, should require a much larger dosage than a normal individual, to produce the constriction effect upon the intestinal unstriated muscle. In the above case histories, it will be noted that this is true in those cases that later reacted to Pituitrin treatment. The writers have also found a positive Pituitrin reaction in many bilobar insufficiencies of the pituitary gland, without polyuria, such as a typical Froelich's disease. In some of these cases it takes two or three times the amount of Pituitrin required in a normal individual to produce this response. This same "intestinal" reaction is produced by a large amount of Extract of the Posterior Lobe Pituitary by mouth. The oral administration has the advantage of avoiding

an undesirable local reaction, but has the disadvantage of being very expensive because of the large amount required to produce an effect equal to that received by subcutaneous use. The "vascular" reaction consists of transient pallor, usually appearing within three to four minutes after hypodermic administration and rarely lasting more than five minutes. A prolongation of this "vascular" reaction, to which are added severe nervous symptoms, has been defined the "general" Pituitrin reaction. The "general" reaction consists of tachycardia, syncope, and extreme emotionalism, associated with prolonged pallor. This reaction is considered a toxic or untoward one and should be avoided when administering this substance. It is rarely the result of a smaller dose than that which produces the intestinal reaction, but should it occur before the intestinal reaction, the indications would be to decrease the size of the dosage and not to attempt to give one sufficient to produce the intestinal response. The occurrence of the "vascular" reaction should caution one to observe the patient for a short time to see that this effect passes away without additional signs of tachycardia, syncope, or emotionalism. In order to do this, the patient receiving Pituitrin should be detained under observation for at least fifteen or twenty minutes after injection, so as to determine what reaction occurs. If the vascular reaction of pallor alone occurs and disappears without additional signs of general reaction, the patient may be dismissed without danger of this untoward and alarming reaction occurring later. If, after giving an injection of this substance, however, the vascular reaction, in the form of extreme pallor, persists, there is likelihood that other signs of the general reaction will occur. If the vascular reaction lasts longer than a few minutes, the patient should not be allowed to pass out from under personal observation, for fear that serious symptoms will occur, which in certain instances might even lead to disastrous results, such as cardiac or cerebral accident. With this knowledge of the possible toxic effects of Pituitrin as a guide to their prevention, one is safe in administering this medication.

The amounts of Pituitrin producing these different reactions should be noted, so as to help determine the amount of insufficiency of this gland, as well as to accurately measure the dose necessary to overcome this hypophyseal deficiency. It has been the writers' custom in adults to begin with a dose of Pituitrin (0) minims 5, and



gradually increase it 1 minim each day until the "intestinal" reaction is produced. This dosage is then considered the tolerant physiological dose sufficient to overcome the insufficiency of the gland. In infants and children, 1 minim, or, if below one year of age, a fraction of a minim, is used as the initial dose. When this extract is given by mouth, the adult is started with minims 15 to 20 a day, and the dosage increased minims 2 to 3 each day until the "intestinal" reaction above described is obtained. This dosage is then continued, and careful observations made upon the polyuria, headaches, nervous disturbances, etc., for a number of weeks, to determine its effect upon the symptomatology. If, after two or three weeks of intensive treatment, with a carefully measured tolerant dose, there has been no decided effect upon the chief complaints, the case is considered one not reacting to treatment. Besides the hypodermic administration of Pituitrin extract, the entire gland substance is given by mouth, Pituitary Substance (Parke, Davis & Co.), in dosage of grains 10 to 30, after meals. The writers have never observed "intestinal," "vascular," or "general" reactions from the oral administration of this substance. It is understood that substitution of Pituitrin or Pituitary Substance, as described, is indicated only in those cases in which there is definite hyposecretion or insufficiency of the infundibulum or the posterior lobe of the hypophysis.

In those cases in which this syndrome is associated with the opposite activity, *i.e.*, a hypersecretion or overfunction of the gland, this substitution treatment would be contraindicated. Pituitrin treatment in such cases would undoubtedly aggravate all symptoms, as in Case XI, and consequently might be dangerous. For this reason it is well to first carefully note the effect of even smaller doses of this treatment. If there is any definite increase in polyuria, headaches, or nervous symptoms, treatment should be interrupted at once and given in much smaller doses under careful observation, in order to confirm such reaction. If it can be established that there is definite hypersecretion present, the treatment which would be indicated is one calculated to reduce the already increased secretion and oversupply of this substance to the body. The only agents which are available in these conditions are (1) X-ray, (2) radium, (3) glandular substances physiologically antagonistic to the hypophysis, and (4) surgical procedures. As regards the efficacy of X-ray treatment in hypophyseal

conditions, Schaefer<sup>66</sup> reports 8 cases of tumor of the hypophysis, with acromegaly, treated by this means, 6 showing definite improvement. He accentuates the fact that the head should be so placed that the hypophysis is situated in the focus of the rays. Webster<sup>67</sup> reports a case of early acromegaly who was given sixteen treatments with hard filtered rays, through the temporal and frontotemporal areas, with good results. The first eleven of these treatments were given at weekly intervals. Jangeas<sup>68</sup> was successful in the use of X-ray treatment in tumors and hyperactivities of the hypophysis, stating that the change in the visual fields parallels the benefit and forms a gauge to the efficacy of treatment. Cavazzini-Bergamo<sup>69</sup> reports 2 cases of hypophyseal tumor with acromegaly which were greatly benefited by X-ray treatments by the Beclere method—deep radiotherapy through the two temporal and frontal regions, at first every week, later less frequently. Case X, of the writers' series, did not react to X-ray therapy. As regards the use of radium, theoretically this form of treatment should be possible, either by external or suitable endonasal or endopharyngeal application. We have no clinical data concerning the efficacy of this form of treatment. Certain investigators have ascribed to certain glandular preparations, notably, Extract of Placenta, anti-hypophyseal effects. The earlier results in this field of endeavor have been unfruitful. Surgical procedure is a well recognized method of treatment, with some argument concerning the best method of approach, each technical modification having its own adherents.

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# Industrial Medicine

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## INDUSTRIAL SURGICAL CLINICS

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### SACRO-ILIAC STRAINS AND SUBLUXATIONS

CASE I.—M. B., age thirty-eight, miner.

*History of Present Condition.*—While lifting a chunk of coal one and one-half years ago, onto a high car, this man suddenly twisted his body from the right to the left and had a sudden sharp pain in his lower back and down the left leg. This pain was so acute that he had to be carried home and put to bed, where he remained for several weeks with no treatment except local liniments. A diagnosis was made of lumbar muscle strain. He had severe pain for about a month, according to his own statements, in the left side, which he localizes now to his left sacro-iliac joint. After this the pain was only a dull ache and he several times has tried to return to work in the mines. Each time, upon stooping to lift, he had the same severe pain in this region and down his left leg. He has had about ten months' treatment of various kinds and in the hands of one doctor received very intelligent treatment and was provided with a Goldthwait belt modification, but this gave him very little relief.

*Examination.*—Examination one year after the injury showed that the lumbar muscles were boarded, that he had a limitation of lateral curve of the spine when leaning away from the affected side, that his forward spinal flexion was normal, but he complained of pain in the left sacro-iliac on returning extension, that his left leg flexion on the abdomen was painful, that there was some pain over the exit of the sciatic nerve from the pelvis (Figs. 1 and 2).

*Laboratory examination* showed the urine negative for albumin and sugar. Blood count was negative except that he had a ten-thousand white count. His teeth and tonsils were badly infected.



*Treatment.*—The infected teeth were extracted and the tonsils removed. He was given the usual manipulation and again fitted with a Goldthwait belt, daily bakings and massage, and has been showing gradual improvement.

After a year of this condition the X-ray shows a left sacro-iliac arthritis which is seen in the X-ray as a blurring of the joint margins, as contrasted with those of the opposite side.

CASE II.—B. V., age forty, miner.

*History of Present Condition.*—While lifting a piece of coal to load it onto a car, his right foot slipped from loose dirt on which he was standing, and his body was twisted in a manner that he does not describe definitely; but he had a sudden, sharp pain in his lower lumbar region and had to be carried home.

*Examination.*—He was seen one week after this accident and upon examination showed definitely localized tenderness in the right sacro-iliac joint. He could not move his right leg or his spine in any position without causing acute pain in this joint. He had been in bed since the accident, suffering greatly. His condition had been diagnosed correctly by the local doctor, but no treatment except adhesive strapping over the affected joint had been given.

*Treatment.*—The usual manipulation was performed under an anesthetic and he was fitted with a temporary strap and later with a Goldthwait belt with a pad over the sacrum. In two weeks he was allowed to get out of bed and given daily baking and massage. He returned to work in three months after his original injury and is now doing his usual duty as a miner, which throws the maximum amount of strain upon this joint, of any occupation of which we know.

*Comments.*—These two cases illustrate the same conditions: one case not treated and the other case treated soon after the time of injury. The influence of focal infections upon this condition is also illustrated.

Until recent years it has been generally accepted that the sacro-iliac joint was an immovable articulation between the ilium and sacrum, but this is now a point of great dispute among orthopedists and anatomists. The obstetricians, however, have definitely proved that the pelvic outlet can be increased or decreased in size at term in pregnancy by either the hyperextension or hyperflexion of the thighs on the body. Litzenberg, of Minneapolis, has shown by careful measurement that pubiotomies may be avoided in many instances by

**Fig. 1.**



**Right sacro-iliac arthritis. Note full lateral curve of the spine, patient bending towards the affected side.**

**Fig. 2.**



**Right sacro-iliac arthritis. Note limited lumbar curve with fixation of the lower segment when patient leans away from the affected side, putting tension on the right lumbar group which are in spasm on account of inflammation in the right sacro-iliac joint.**

FIG. 3.



First motion in the Magnuson method of reduction; flexion of the straight leg on the abdomen to a right angle or better. The knee of the opposite side must be held to the table in this part of the manipulation.

FIG. 4.



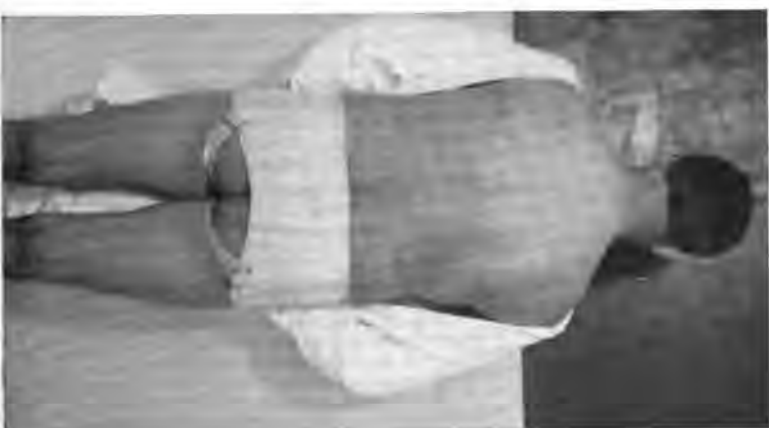
Second part of the manipulation. Leg hyperextended over the edge of the operating table. Patient's shoulders must be held on the operating table by an assistant, in this manipulation, lest the body come into line with the leg. The weight of the limb alone is usually sufficient to bring this full extension while patient is under a gas anæsthetic. Force must not be used to any great extent or the ligaments around the hip-joint will suffer.

Fig. 5.



(operator's hands under the crests of the ilium. Patient is lifted with a quick sharp pull from the operating table, and the lumbar curve held exaggerated while an assistant places a small pillow between the patient's lumbar spine and the operating table. Nitrous oxide gas is used to obtain partial relaxation and analgesia in all cases.

Fig. 6.



Goldthwaite belt applied between the crests of the ilium and trochanters and buckled in front, giving a circular pelvic support.

FIG. 7.



Over-zealous splinting. X-ray by Dr. H. E. Potter, showing fracture of ulna 3 inches above the wrist.

governing the inlet and outlet on this principle, and yet his colleague, Erdman, from an anatomist's standpoint, claims that the ilio-sacral joint is absolutely a fixed joint and contains only a few patches of true synovial membrane. These men are probably both correct. In a normal, healthy joint there is probably no motion between the bones, or such slight motion that it is negligible; but in a relaxed condition of the ligaments there is motion of the bones, and this relaxed condition can be brought about by traumatism, overstrain, or general debility as easily as in pregnancy, and more quickly.

We know that ligamentous tone increases or decreases as the general health or circulation of the individual indicates.

The sacrum is set in between the ilii as the inverted keystone of an arch; that is, with the wider part below and the narrower part above. The entire weight of the body above this joint presses downward and forward on the sacrum, and any force driving upward is transmitted directly to this joint through the legs and hips, so there is a constant tendency to force the sacrum downward and forward and the ilium upward and backward. Also when in a stooping position, or when sagged down in a seat with the feet extending straight forward, the lumbar curve disappears. With the point of greatest leverage at this joint, the weight of the body is thrown on the tuberosities of the ischium, pressing them forward, and consequently, these being in the lower part of the pelvis, have a tendency to throw the upper part of the ilium backward. The weight of the upper body around the shoulders is usually resting against the back of the seat, and we have here a lever with its fulcrum falling in the upper part of the sacro-iliac joint, and the weight around the abdomen tending to be forced backward and downward until it reaches a support.

This same mechanical condition of strain exists when the individual bends forward to pick something off the ground or lift something, except that this motion is a more acute effort. Which one of us has not seen patient after patient have a "crick" in the back after having stooped over to pick something off the ground or lace his shoes, or which one of us has not seen patient after patient come in complaining of chronic backache after leaning over, digging, washing, etc., for a considerable period of time? It is a common experience after long drives in a carriage or automobile, sagged down in the seat with only support at the shoulders and ischial tuberosities and

with the lumbar curve flattened out, to have an extreme stiffness or soreness in the sacro-iliac region as well as in the lumbar region.

These mechanical points are important in the diagnosis and treatment of various forms of backache, and unless the medical profession wakes up to the fact that our bodies are built on mechanical principles and that many things that we have groped in the dark about are due to a mechanical fault of one or another, we are doing our patients grave injustice, neglecting our duties as physicians and are allowing those, who are little better prepared to practice medicine than mechanics, to make cures where we have failed, not because they are acquainted with pathology but because some one has appreciated for them the fact that there may be mechanical displacements in the body which give severe symptoms and which can be rectified by mechanical means. These individuals will proceed along one line for every disease because it is the only line in which they have been trained; and we, in the power of our supposed knowledge, are inclined to sneer at something we know nothing about and which we have made no study of, and of which, therefore, we cannot appreciate the merits.

Let us look at the sacro-iliac joint from an anatomical standpoint. It is the inverted keystone of the pelvic arch which transmits every shock between the upper and lower parts of the body. It is held by strong ligaments posteriorly, running in various directions between the ilium and sacrum, the erector-spini muscles being attached about its posterior upper surface. The lower and internal surface is also held in place by strong ligaments and over these ligaments lies the sacral plexus which unites to form in the main the great sciatic nerve.

Granted a relaxation of these ligaments due to any cause, such as overexposure to cold, overlifting, auto-intoxication, etc. With the leverage allowed by the long upper part of the body and the longer lower part of the body the muscles of the legs being attached entirely to the pelvis, and granted a stooping posture, the hamstring muscles pull tightly on the ischial tuberosities, holding the ilii in definite relation to the femurs in the extreme flexed position. The flexion continued, the entire weight of the flexion tends to throw the sacrum forward, and if the ilium is held fixed by the hamstrings attached about the lower part of the pelvis, the tendency of the upper part is to come into line and be forced backwards. We, therefore, have an overstrain thrown on the ligaments, which for some cause or other

are relaxed, there is a quick, sharp pain in the back, and upon attempt to straighten up the individual finds it impossible. His lumbar curve has partially disappeared. He is forced to support his body by placing his hands on the affected side, to drag his leg after him, to ease his weight into a chair, resting the weight on the unaffected side, and he has produced a slight subluxation of the upper part of the sacro-iliac joint with a resultant overtension on the ligaments, which immediately causes excruciating pain and which he calls a "crick" in the back, or lumbago.

It is true that a very small minority of X-ray pictures of these things show the actual results grossly enough for one to be sure of the pathology, and, therefore, it is hard to prove, but viewed from the standpoint of mechanics, it should be easy enough to see. If this subluxation is replaced by a manipulation of the leg, the pain will disappear immediately, exactly as it came, and the patient who one minute was not able to walk with comfort or sit down squarely, and must drag one of his limbs behind him, the next minute is free from pain and able to perform any function which he had performed before the accident, except lift heavy weights. This is a strong statement, but absolutely true, as proved by those cases which can be produced and authenticated.

If the subluxation is not replaced, the patient is forced to go to bed applying liniments, heat and massage until the ligament which is overstrained adjusts itself to its new position or the joint automatically replaces itself on account of some motion which the patient performs. This, however, is only the acute class. In some cases the pain in the back will persist, gradually extending down the leg over the course of the sciatic nerve. This is due to an extension of the traumatic inflammation set up in the ligaments around the internal anterior surface of the joint by the continued overstrain of the ligaments. The inflammation extends to the sacral plexus which lies immediately upon the anterior surface of the joint. This pain down the leg usually does not come on for about ten days to two weeks from the beginning of the ligamentous pain in the back, but it has been diagnosed sciatica and sciatic rheumatism since the days the medical profession first recognized the sciatic nerve.

An acute tension on the sacral plexus will give the same symptoms more acutely, and cases which have been diagnosed sciatica by



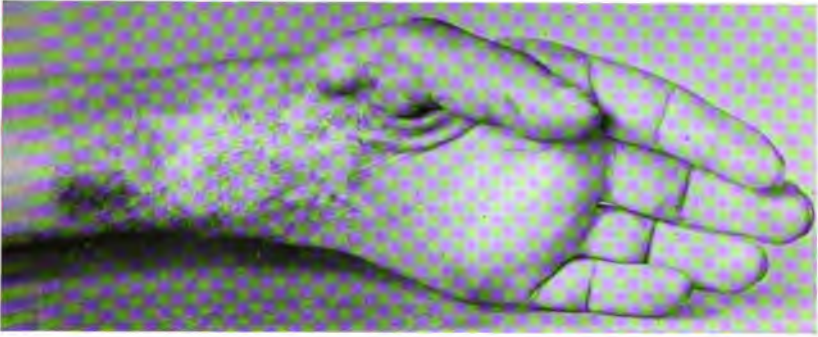
expert internists and have been subjected to the usual forms of treatment for a period of two weeks without result, have been cleared up in the twinkling of an eye, not to return again, after the proper manipulation. The manipulation used by us for the reductions of the sacro-iliac joint is as follows:

The leg, with the knee straight, is brought to a right angle with the body, the patient lying on his back on the operating table (Fig. 3). This tightens the hamstring muscles which run from the tuberosity to their attachments at the back of the knee. This motion pulls the lower part of the pelvis forward and the upper part of the pelvis backward and puts the patient in the same position in which he usually produces the subluxation. In other words, this increases the deformity and unlocks the joint. When the muscles are tight and the patient is either not expecting any other motion, or is under an anæsthetic, which is best in most cases, the leg is quickly brought into hyperextension over the edge of the table (Fig. 4). This in turn pulls on the "Y" ligament and the tensor fascia lata, which are attached to the ilium above and in front, and when the leg is brought into extension are pulled tight, pulling the upper part of the pelvis forward, and then with the leg still in full extension over the edge of the table, the operator's hands are placed under the junction of the ilium and sacrum and the patient lifted from the operating table with a quick, sharp jerk which increases the curve in the lumbar spine, and while supported thus by the operator an assistant places a pillow between the patient's lumbar curve and the operating table (Fig. 5).

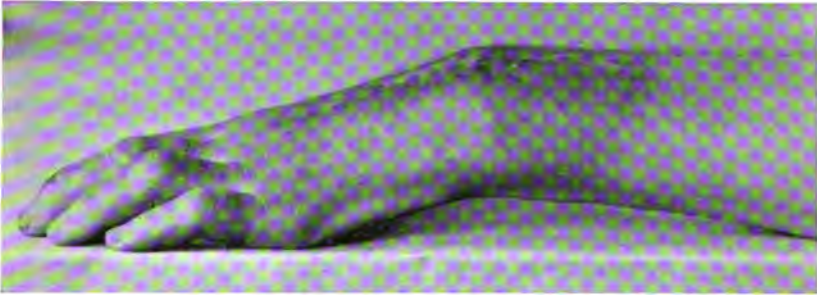
Gas anæsthetic is practically always used in these reductions in order to get a certain amount of relaxation. If gas anæsthesia is not used an assistant must be present to hold the patient's opposite knee flat on the table in the first motion and to hold the patient's shoulders on the table by resting weight on the chest in the second motion. Otherwise, the discomfort produced by manipulation will cause the patient to draw up the opposite leg and when the second manipulation is performed he will raise off the table to a sitting position, thereby preventing any overextension on the injured leg.

The patient is helped off the operating table after this manipulation and circular bands of adhesive plaster are put around the pelvis, starting at the junction of the sacrum and the fifth lumbar and con-

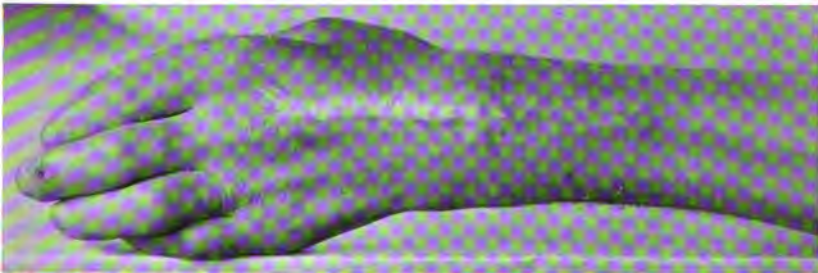
**FIG. 8.**



**FIG. 9.**



**FIG. 10.**



**FIGS. 8, 9 and 10.—Pressure atrophy of skin and fascia involving both flexor and extensor tendons and resulting in total loss of use of the hand due to tight splinting of arm with splints left on for two weeks without inspection.**

**FIG. 11.**



**Fracture dislocation of the scaphoid and first cuneiform. Showing the ununited fracture of the inner projection of the scaphoid.**

**FIG. 12.**



**Fracture dislocation of the scaphoid and first cuneiform. Lateral view showing forward displacement of scaphoid and first cuneiform bones. X-ray by Dr. H. E. Potter.**



FIG. 13.



X-ray showing fracture of upper end of right tibia and fibula, comminuted, fracture of tibia extending into the knee joint opposite the intercondyloid notch of the femur and not into one of the articular surfaces.

tinuing down to the end of the sacrum. About four 3-inch strips of adhesive plaster, overlapping on the edges, are all that will be found necessary. These must be put on snugly and allowed to stay until proper brace or belt can be fitted. We use a Goldthwait belt with a posterior pad as shown in Fig. 6.

#### OVERZEALOUS SPLINTING

J. L., age thirty-four, carpenter.

*History of Present Condition.*—While riding in an automobile he was involved in a collision and his left ulna was fractured about three inches above the wrist (Fig. 7). The arm was set on the same day and put up in board splints, anterior and posterior, down to the base of the fingers. The boards were bandaged on snugly and were not removed for two weeks, in spite of the fact that the patient complained of pain and numbness and finally of a bad odor from the arm. When the splints were finally removed, it was found that there was an ulcer about four inches long over the back of the hand and wrist and another over the anterior surface of the wrist, its centre being about over the trapezium. He says that his fingers were numb continually and that he had very slight motion in them. The doctor told him to leave the splints on as it was necessary to have them tight.

*Examination* (August 30, 1920).—Examination shows the fingers of the left hand fixed in extension at the metacarpal-phalangeal joint and in flexion at the middle interphalangeal joint. There is such slight motion of the fingers that it is almost negligible. Upon forced flexion it is found that the extensor tendons are firmly bound by adhesions to both the superficial and underlying structures. The long and short flexor to the thumb has entirely sloughed away so that there is no motion in this member whatsoever. Over the trapezium and the surrounding area there is an ulcer about the size of a half dollar which has been apparently deep, and the bundle of flexor tendons in this region is fixed firmly by adhesions and adherent under the annular ligament. There is no sensation in the back of the hand; a pin can be thrust in without patient giving any evidence of sensation whatsoever. There is no sensation in any part of the hand supplied by the ulnar or median nerves, the only sensation being located over the back of the thumb, which is supplied by the external radial.

*Comments.*—It is hardly necessary to make any comments on this case. To the young surgeon who has not learned by bitter experience that swelling may take place under a splint and do terrific damage in

an extremely short time, it may be well to call attention to the fact that it is not necessary to have this fixation of plaster, but any constricting force or pressure will result in the same damage as will a circular plaster cast which is put on too tightly. This, of course, is the exceptional case, but we see so many Volkmann's ischæmic contractures or paralyses due to the same sort of splinting, and the damage is done in a few hours and not in days, that any immobilized member should be under constant observation until all danger of swelling beneath the immobilization dressing is past.

In this case the surgeon had all the signals of distress: pain, numbness, inability to move the member, and finally the odor, and he paid no attention to any of them in spite of the fact that he is an individual who, I am sure, knows the meaning of these signs, if he but stopped to think. Every individual who has to do with injuries which are immobilized by fixation dressings, should be warned of the rapidity with which the damage in these cases can be done, and be on guard for these accidents. This injury should have resulted in no permanent disability. As a matter of fact, it has resulted in the permanent total loss of use of a hand, in a highly paid, young wage-earner, and the responsibility of an accident of this kind should weigh heavily on the minds of those involved.

#### FRACTURE DISLOCATION OF THE SCAPHOID AND FIRST CUNEIFORM

O. L., age thirty, laborer.

*History of Present Condition.*—This man fell from a smokestack about forty feet high, on which he was working, landing on a slanting surface with his toes pointing downward, so that his weight was thrust sharply forward, transmitted through the metatarsal bones.

*Examination* (May 28, 1920).—He now has a marked bulging on the dorsum of the foot which feels like a dislocation between the tarsus and metatarsus of the second, third, and fourth. The plantar fascia is relaxed and he complains of pain across the instep. The scaphoid attachments are tender and there is tenderness on the bottom of the foot opposite the bulging.

*X-ray* (Figs. 11 and 12) by Dr. H. E. Potter, whose report is quoted, shows "evidence of an old fracture dislocation, involving several of the tarsal bones and a fracture of the fourth metatarsal near the distal end, now perfectly healed. In the tarsus the scaphoid and

first cuneiform bone are seen to lie forward from their previous position, nearly half an inch. Various lines of fracture may be seen through the complex of other cuneiform bones near the cuboid. There is also an ununited fracture of the inner projection of the scaphoid. Of these, the most pronounced change is the displacement of the scaphoid and first cuneiform."

*Comments.*—This is an extremely unusual and interesting case because of the peculiar mechanism, the weight being driven forward exactly through the long axis of the metatarsal bones. The shaft of the metatarsals held for the most part and the large bones above, the astragalus and tibia, caught the scaphoid and first cuneiform between the transmitted force from below and the transmitted weight from above, and squeezed them out and up like a pea out of a pod. This, of course, shortened the foot from front to back and removed the keystone of the arch of the foot which is the scaphoid, pressing it upward so that it no longer acts as the keystone of the arch. There should have been some attempt made at the time of the injury to force the scaphoid and cuneiform back to their normal positions, by pressure from above, an attempted moulding of the foot with inversion maintained by a plaster cast. On account of the swelling, however, there was no such attempt made and by the time the swelling subsided, the bones were so fixed in their displaced position that it was impossible to replace them.

This patient was estimated to have a 60 per cent. disability of the foot on account of this injury, because of the breaking of the longitudinal arch, displacement of the bones and the consequent inability of the patient to walk or stand for any considerable length of time. There is no treatment at this time, so far as we know, which will do any permanent good.

#### ARTHRITIS OF THE INFERIOR RADIO-ULNAR JOINT

B. M., age forty.

*History of Present Condition.*—In November, 1918, patient was pushing boxes down a skid. She had her left hand on a pile of boxes with her elbow out in back and her forearm horizontal, when another pile of boxes struck her elbow, giving her wrist a sudden sharp twist and impact. There were no bones fractured but the carpus, acting as a wedge, forced the radius and ulna apart, tearing at least part of the fibers of the radio-ulnar joint.



*Examination* (September 12, 1919).—She complains now of pain in this joint when rotating the forearm, but there is no pain on passive or active flexion and extension of the wrist, nor is there any pain or tenderness in the carpal articulations. Pressure made on the radius and ulna, tending to force the joint surfaces together, gives severe pain in the joint, whether the pressure is made above or at the level of the joint. A crepitation in this joint may be felt as the patient rotates the forearm. With both hands resting prone on the table, a comparison of the movements in the distal ends of the two ulnas was made, pressing them downward towards the flexor surfaces with the thumbs of the examiner's hands. *The ulna on the injured side could be pressed downward towards the flexor surface for fully half an inch more than the one on the opposite side*, and when the pressure was removed, would slip back and become more prominent than the one on the uninjured side. From this position of full pronation, there was 50 per cent. rotation to full supination. This was, of course, caused from a backward or extensor slipping of the distal end of the ulna so that the radius did not revolve in its full arc over the articular surface, the ulna being too far back.

*X-ray* taken by Dr. N. E. Potter showed no signs of fracture or dislocation, all structures appearing normal and in apposition. This is easily explained because the ulna and radius fit together at this point in a curved line from the front to back and in order to show any appreciable separation of this joint, the bones would have to be apart far enough to let the rays pass directly through, there being no overlap of the radius and the ulna. If we could throw the rays in the long axis of the arm so that they would show a perfect cross-section of the wrist, no doubt they would show a separation of this joint, but neither from a lateral or antero-posterior view could this separation be shown unless it was extremely great.

*Comments.*—There was no treatment instituted in this case except bandaging of the wrist. If, at the time of the injury, the ulna had been forced forward, the hand brought into full supination, and the arm placed in a cast with the elbow flexed to hold the arm in proper position, this injury would undoubtedly have healed and given no serious disability. As a matter of fact, the author has treated other cases which have gone over a period of a number of months in this way, and obtained good results.

It will be apparent that full supination cannot be maintained without the cast extending over the flexed elbow, inasmuch as there would be nothing to hold in the lateral plane, the axis of both bones of the forearm being parallel, in supination, the radius rotating around the ulna to reach pronation, it is evident that the cast must extend over the flexed elbow and up the arm in order to maintain the full supination.

At this time there has been set up an arthritis in this joint which is seriously disabling the patient and which will give her a certain percentage of permanent disability, whereas, with proper treatment, she should have had none.

#### FRACTURE OF BOTH BONES OF THE LEG EXTENDING INTO THE KNEE-JOINT

D. G., age thirty-one.

*History of Present Condition.*—On March 1, 1920, this patient was on top of a telegraph pole with his leg around the pole, cutting wire. As he cut the last wire, the pole broke at its base and fell. His right leg was caught under the pole as it struck the ground. He received a fracture by direct violence of the upper end of the tibia and fibula, oblique and extending into the knee-joint, about its centre. There was immediate and great swelling, which when aspirated, showed thick blood in the knee-joint with fat globules present. The swelling was so great it was treated for the first three weeks in a fracture box, with ice bags to reduce. For the next three weeks with a plaster cast with the knee straight, no attempt being made to establish motion either passively or actively during this time.

*Examination* (April 16, 1920).—When the patient was examined by us it was found that there was practically no motion in the knee, that the fragments were in very fair apposition, but there was still motion between them. There was some swelling and induration in the knee-joint and the whole leg was painful. Cast was removed and a half cast applied over the anterior surface of the leg from just below the knee to the ankle, to give support, but allow motion in the knee. Dry heat was applied for half an hour three times a day and gradual massage, especially directed at the knee-joint, was given for an hour a day, the object being to promote absorption of the induration, relieve the swelling, and reestablish the elasticity in the ligaments by careful, graduated motion. Patient was encouraged to actively exercise the joint. Resistive exercises were started as soon as there was ten degrees of motion present in the joint, and in two

weeks from the time of the first examination the patient was encouraged to use the leg in walking, it being estimated that there was enough union at that time, although not perfectly solid, to bear the man's weight with the assistance of the anterior splint, with the idea of promoting callus formation by use. In one month from the date of examination ninety degrees of motion had been established.

*Comments.*—It was a fortunate occurrence that this fracture, extending into the knee-joint, broke through opposite the intercondyloid notch of the femur. Therefore, a more favorable prognosis could be made than had the fracture extended into one of the articular surfaces where there was heavy weight-bearing. In fractures into any weight-bearing joint where the fracture extends into the joint at a point where the greatest weight falls on this joint, a very serious disability can be expected, since any slight displacement of fragments leaves a rough surface and therefore a painful joint. This is not so true, of course, in the joints of the arm where there is no weight-bearing and is more especially true of the knee-joint and of the ankle-joint than any others. Where there is callus formation in a joint that leaves a rough surface, the opposing cartilage is lacerated whenever motion occurs which gives severe pain.

Some comment is due the type of fluid aspirated from this joint. The thick, bloody material signifies only that there is a blood-vessel torn, which bleeds into the joint, and this blood is mixed with joint fluid, but when globules of fat are found floating in this fluid, it can be definitely said, without X-ray, that there is a fracture into the joint which has allowed fat to escape from the internal bone substance. This is an important diagnostic point in differentiating between a fracture into the joint and a torn ligament without the assistance of X-ray. Even in the presence of X-ray, there are numerous occasions where in a large joint, like the knee, the fracture line may be in several planes, so that the X-rays will not pass through, showing a fracture and unless the irregular outline can be discovered on the joint surface, the fracture may be overlooked. If one is suspected, skiagrams at various angles should be taken in order to determine whether there are any irregularities along the smooth line of the joint.

In case of all joint fractures, the joint should be mobilized at the earliest possible moment. It has been plainly shown by reports of

cases occurring during the recent war, that motion does not have to be through a great number of degrees, but it must be often repeated and preferably active. In infected joints it is almost essential that it should be active rather than passive, and it should be put squarely up to the patient that it depends on him whether he will have a stiff joint or a movable one. Where there is a fracture with displacement of the fragments on active motion, then the motion should be gentle and passive and controlled by an experienced surgeon or a trained physio-therapist who realizes the dangers of over-movement.

In the original treatment of this case it would have been much better to apply a splint or cast which could have been removed at least above the knee each day and the joint exercised passively, by a carefully trained and experienced operator. We realize that the difficulties of these complicated cases are many and they cannot be simply put up in a cast and allowed to stay there as in olden times, for six to eight weeks, without having some sort of intelligent attention. The training we have all had has made us somewhat fearful of interfering with fractures until we feel that the callus has firmly fixed the fragments in apposition, and it is true that meddling surgery is the cause of considerable non-union, but it is our belief that *it is unintelligent meddling surgery that causes non-unions and not the intelligent use of motion and exercise* (see Fig. 13).

#### PERFORATING INJURY TO THE ULNAR NERVE

J. S., age forty-five.

*History of Present Condition.*—On May 18, 1919, this man was struck by a piece of steel which entered the forearm about four inches above the wrist and just outside of the ulna. There was made a small incised wound, the scar of which is still present. Patient claims that he noticed soon after this injury, at which time he had severe pain, that he had lost strength in the hand, especially the thumb, and that his little finger and part of his ring finger felt numb.

*Examination* (April 12, 1920).—Examination shows a small scar over the outside of the ulna about 4 inches above the wrist, which, when pressed upon, he says gives him pain which runs up to his shoulder and down to the little finger side of the palm of the hand. He has pain in this side of the hand continually and says it is especially bad at night. There is a distinct atrophy of the hypothenar eminence and a very distinct atrophy of the palm of his hand is seen

from the base of the metacarpals to their extremities. There is complete atrophy of the adductors of the thumb. At present there is an abrasion over the outside of the little finger, which he says he received a number of months ago, but which will not heal. There is complete anaesthesia of the little finger and the little finger side of the ring finger (Figs. 14 and 15).

*Comments.*—This is plainly a case of severance of the ulnar nerve by a stab wound, and it should have been recognized by the surgeon that the wound was in the neighborhood of the ulnar nerve. Proper examination should have been made at the time of the injury to ascertain whether the ulnar nerve had been injured, and if so, proper steps taken to suture it, providing there was no infection of the wound at the time. If infection were present, the wound should have been allowed to clean up under proper surgical treatment. In the meantime, the muscles of the hand should have been treated by faradism in the form of Bristow coil treatments, to maintain the tone and strength of the muscles involved, and as soon as possible the severed ends of the nerves should have been brought together by fine catgut sutures, after having been cut with an extremely sharp scalpel, straight across to remove any possible scar tissue involving the ends. If it was necessary to delay the suture for any considerable length of time, then it might become necessary to do a two-stage operation on account of the retraction of the ends of the nerves, putting in a black silk suture, drawing the ends of the nerves as close together as possible, putting the wrist up in hyperextension, and gradually flexing to stretch the nerve to be united at a second operation, in this way bridging the gap which would otherwise put so much tension on the fine sutures which must be used at the time of the nerve union, so that there would be a break in that union.

#### SPONTANEOUS OR PATHOLOGICAL FRACTURE OF THE FEMUR

H. W., age twenty-eight, laborer.

*History of Present Condition.*—While pushing on a brick car containing about a thousand bricks, he felt something snap in his right leg and fell to the ground. He had no pain whatsoever, but when he tried to get up he found that he could not walk. He was sent in an ambulance to the hospital.

Previous medical history shows that he had a chancre eight years ago and had had treatment irregularly for three years thereafter.

FIG. 14.



Ulnar nerve injury. Loss of epidermic sensation inside line—this is loss of light touch—tested by cotton.

FIG. 15.



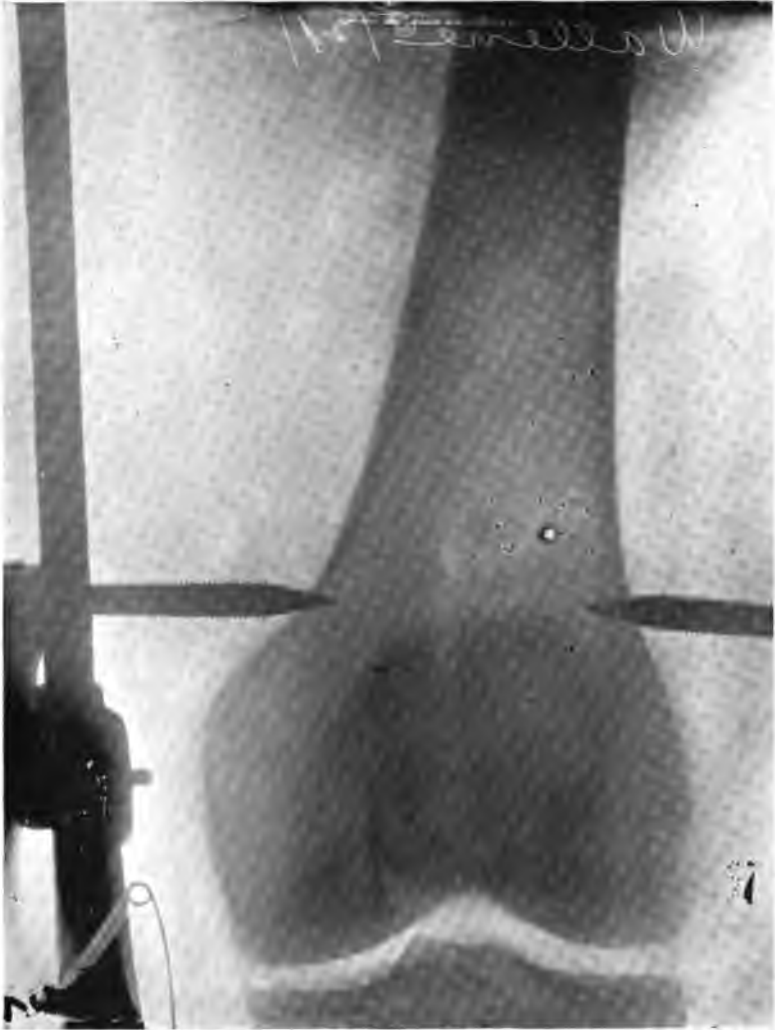
Ulnar nerve injury. Loss of photopathic sensation indicated by dark area—this is loss of recognition of cutaneous pain—tested by pin prick.

**FIG. 16.**



**Pathological fracture of the shaft of the right femur,  
showing overlapping and displacement.**

FIG. 17.



Pathological fracture of the right femur showing position of tongs used for traction.



**FIG. 18.**



**Pathological fracture of femur, taken at the tenth week, showing absolute lack of callus formation.**

*Physical Examination.*—Right leg shows a comminuted fracture of the right femur at the junction of the lower and middle third with considerable overlapping and displacement. Right leg from the anterior superior spine to the internal malleolus is 2 inches shorter than the left (Fig. 16). He has an Argyll-Robertson pupil, absence of knee jerks, has incontinence of urine and feces. Wassermann is four plus. His urine shows heavy trace of albumin, many hyaline and granular casts, no sugar.

*Diagnosis.*—Locomotor ataxia, chronic nephritis and spontaneous or pathological fracture of the right femur, due to syphilitic osteitis.

*Treatment, by Doctor Pond.*—This man was placed in bed with a Hodgkins splint; traction was made by adhesive extension at first, but this did not reduce the deformity, so traction was made with tongs, the position of the tongs as shown in the illustration (Fig. 17). This traction secured an excellent alignment of the fragments as is shown in the third X-ray picture (Fig. 18).

The treatment of syphilis was started at once, but on account of the chronic nephritis, it was only possible to give him small doses of neosalvarsan, and he received .15 of a gram weekly of neosalvarsan during his stay in the hospital. He was also given inunctions of mercury and potassium iodide, 60 grains, in divided doses daily.

At the end of five weeks the X-ray showed practically no callus. At the end of ten weeks the X-ray still showed absolute absence of any callus formation. He was in slightly better physical shape and had better control of urine and feces. At this time the extension was removed and a plaster cast applied with his knee in flexion. His Wassermann was still positive. He now developed some mental symptoms and was more or less continually depressed. The psychiatrist, Doctor Weisbrenner, was called in to see him and diagnosed his condition as general paresis finding a positive spinal fluid Wassermann. He was fitted with a calipered Thomas splint with a leather cuff around the fracture, and sent to the County Hospital for the Insane, where a diagnosis of general paresis was confirmed. About one month after his admission, he was paroled from this hospital in the care of his brother, and admitted to another hospital, where he was operated on for this ununited fracture and died of surgical shock, following the operation.

*Comments.*—First, a question of liability: The insurance com-

pany in this case is claiming that this was not an accident, but that it was due to the syphilitic osteitis and that it might have happened at any time or any place. This, of course, will be subject to contest, as the Supreme Court of Illinois has held that whatever predisposing condition may exist if the employment is the immediate occasion of the injury, it arises out of the employment because it develops within it. When the exertion of the employment acts upon the weakened condition of the body of the employee or upon an employee predisposed to suffer injury in such a way that a personal injury results, the injury may be said to arise out of the employment.

Second, medical examination of employees: This case gives a remarkable illustration of the value of the medical examination of all employees to both the employer and the employee. For the employer in this case it would have saved a great deal of expense, either for a long-continued law proceeding or for the payment of death claim and hospital bills, as certainly any man with the marked and advanced syphilis that this man had should not have been employed as a laborer. Even a casual examination by a layman would have developed the fact that this man had incontinence of urine and feces.

The value to the employee in this case is well known, as the man stated that he thought his syphilis had been cured and did not connect his physical condition with his syphilis of eight years previous. Of course, any young man of twenty-eight years could have been greatly benefited by treatment, which treatment would certainly have been recommended if there had been in this plant the medical examination of employees.

*Treatment.*—The treatment in this case is believed to have been excellent, except that apparently this man was operated on after his parole from the County Insane Asylum, without sufficient laboratory and other physical examination, as certainly an operation for an ununited fracture of the femur would not have been done if his Wassermann had been found to have been four plus.

#### FRACTURE OF LOWER END OF RIGHT RADIUS, UNREDUCIBLE

J. W., age thirty-one, expressman.

*History of Present Condition.*—March 1, 1920, this patient was loading express when a pile of boxes fell, knocking him forward, with his right hand extended; the boxes fell on top of him. He sustained a fracture of the lower end of the right radius and a fracture of the styloid of the ulna. Two attempts

under anæsthetic had been made at the time the author saw him, to reduce this fracture, without avail.

The author made a third attempt under the fluoroscope and again failed, although very strong traction was made. It was decided to make an open reduction which was done on the sixth day following the injury.

*Operation.*—An incision was made laterally over the radius and the fragments exposed. The lower end of the upper fragment was driven downward and forward, the lower fragment overriding it in the usual Colles fracture deformity. The pronator quadratus had a tendency to displace the lower end of the radius towards the ulna and extreme difficulty was encountered in bringing these fragments into apposition. It will be noted in the X-ray (see Fig. 19) that the lower end of the radius is driven upward, and that the distal articulation between the radius and ulna is entirely displaced. A strong leverage between the fragments was necessary to pry them back into position and force the lower end of the radius down into place. An ivory peg was inserted through the outer part of the lower fragment into the medullary cavity of the upper fragment to offset the displacing power of the pronator quadratus (see Fig. 20). The arm was put up in a posterior splint, held in the usual downward and outward position, with slight flexion of the wrist commonly used in Colles fractures. The stitches were removed on the seventh day and passive motion instituted. This was repeated daily with exercises of the fingers and wrist, so that in six weeks from the date of the operation the patient had not only union sufficient to remove all support, but no atrophy of the arm and no stiffness of the fingers or wrist. In the ordinary Colles fracture, these results can be obtained in four weeks, inasmuch as there is little or no tendency to displacement of the fragments, once properly placed in apposition, and Stevens believes that it is not necessary to have any fixation dressing on for more than a week. There is one fact firmly established, however, and that is that fractures into or near joints must be mobilized early if satisfactory results are to be obtained.

**FRACTURE OF THE SHAFT OF THE FEMUR, ILLUSTRATING VALUE OF  
LATERAL TRACTION TO CORRECT DEFORMITY**

D. L., age twenty-five.

*History of Present Condition.*—Six weeks prior to examination this young man was working in a mine, when he fell off a car and fractured the right femur about three inches above the knee. He was sent at once to a local

hospital where Buck's extension was applied for two weeks. He was then put up in a cast. This cast was removed at the end of the sixth week, at which time an X-ray was taken, the result as shown in Fig. 21.

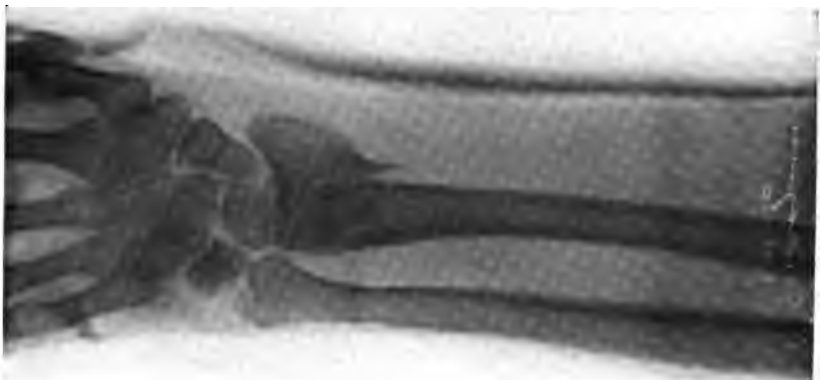
*Examination.*—The right femur from the anterior superior spine to the internal malleolus was  $1\frac{1}{2}$  inches shorter than the left. The X-ray shows that the main deformity is due to the angulation, the apex of the angle outward. The patient complained of great pain at the site of the fracture on any motion of his knee-joint.

*Laboratory Examination* showed his Wassermann to be negative, urine negative for albumin and sugar. There were no focal infections; teeth were in good condition, tonsils not enlarged.

*Treatment.*—In a man of this age it is absolutely not to be considered to leave his leg  $1\frac{1}{2}$  inches shorter than the other one. Open operation was considered, but upon careful study of the X-ray and a clinical study, it was decided that the callus about the fragments was still soft enough to permit a correction of this deformity with extension. Therefore, the young man was placed in a fracture bed with Buck's extension applied from the knee downward, with a traction of two bricks, nine pounds. A transverse traction was also made by a band of adhesive 6 inches wide with a pad placed over the outside of the leg at the apex of the angulation. This band of adhesive fitted over this pad and over the angulation, and the extension was made toward the opposite leg. The weight was one brick, or half the longitudinal extension. At the end of three weeks this extension was removed and it was found that the shortening had been reduced to  $\frac{3}{8}$  inch. The X-ray pictures showed that the reduction in the shortening was due to the reduction in the angulation, as the position of the ends of the bones, so far as overlapping was concerned, remained practically the same. The fragments were now in direct line with each other, giving a straight weight-bearing line. The young man had no pain at the site of fracture and after a short period of time had no pain on motion of his knee-joint. He was allowed to return to his home with the heel of the shoe on the affected side slightly raised to correct the remaining slight shortening.

*Comments.*—In this case the cast that was applied after the original extension had been removed, was not of sufficient thickness and was not snug enough to prevent a return of the original deformity. The cast consisted only of a few layers of adhesive plaster bandage.

**Fig. 19**



**Fracture lower end right radius and fracture of the styloid process of the ulna.**

**Fig. 20.**



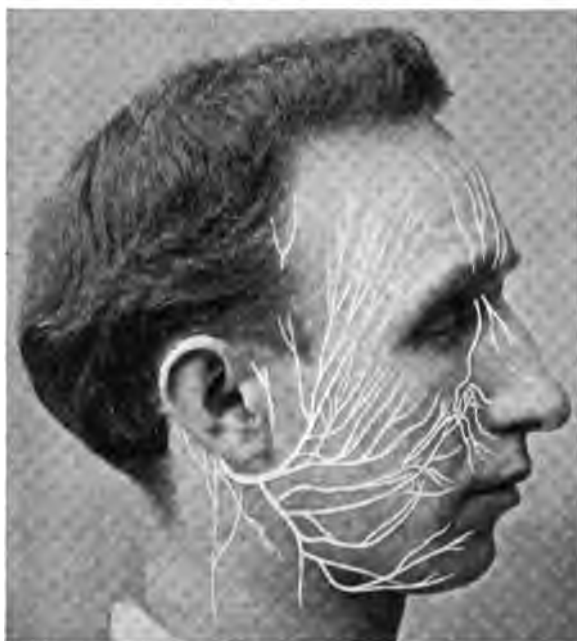
**See ivory peg inserted through outer part of lower fragment into medullary cavity of upper fragment.**

**FIG. 21.**



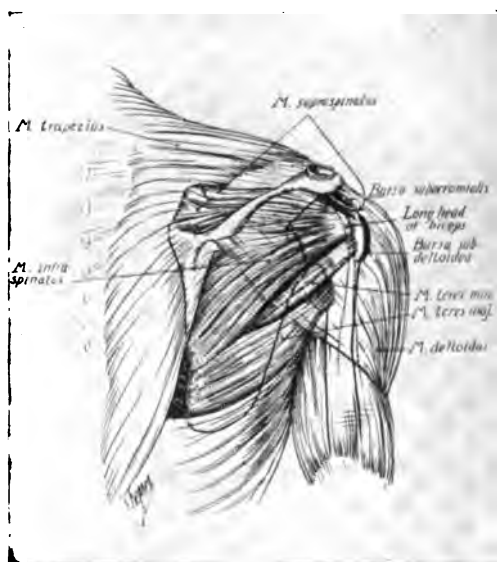
**Fracture shaft of femur.**

FIG. 22.



Showing distribution of facial nerve.

FIG. 23.



Showing bursæ of shoulder and muscles.



FIG. 24.



Clearly modification of aeroplane splint showing adjustments and positions. No. 8 shows side view with nearly complete external rotation.

This case illustrates the value of traction applied even as late as six weeks, to correct angulation and deformities in fractures of considerable duration. In this case it saved the young man an open operation, it lengthened his leg  $1\frac{1}{8}$  inches, it reduced an angulation that would have been impossible for him to use to carry his weight, and gave him a correct weight-bearing line in his fractured femur.

#### FRACTURE OF THE JAW, OPERATED, WITH DISASTROUS RESULTS

L. F. S., age twenty-eight, miner.

*History of Present Condition.*—February 28, 1918, this patient, while working in a coal mine, had his head caught between a load of coal and the roof. He sustained a simple fracture of the ramus of the right inferior maxilla, with no injury involving the nerves. It was deemed impossible by the attending physician to hold the fracture in place and the patient was operated and the fragments wired with heavy silver wire. The incision was made about an inch in front of the ear and perpendicularly, and he states that when he recovered from the anæsthetic he noticed he was unable to close his right eye.

*Present Examination* (July 12, 1920).—The first thing that he complains of is inability to close his teeth in such a manner that he can masticate his food. There is some angulation of the lower maxilla to the right, and the only two teeth which come together are the two posterior molars on the left side, the incisor teeth not meeting within  $\frac{1}{4}$  inch. Therefore, the upper and lower set, of course, are not parallel, aside from the right deviation. Also he complains of his inability to close his right eye, and constant overflow of lachrymal secretion. He also has constant pain in the right side of the jaw, especially when he opens and closes his mouth.

In view of the fact that there were two serious conditions here, the patient was referred to Dr. Herbert A. Potts for opinion as to what could be done for the relief of the displaced fragments, with the idea of bringing the jaws into better apposition, and the following report was received: "The X-ray shows no evidence of fracture on the left side. The right was fractured at the ramus, the lower jaw is wired in bad position at the base of the neck. A cast was made of his teeth and I find that occlusion can be perfectly restored if we resect the right ramus, possibly at the site of fracture. The pain and tenderness at the site of fracture is due, in my opinion, to the wires which are a source of irritation, and these should be removed. If, during the course of the operation, the seventh nerve can be found, an

attempt can be made to repair it. It will not be advisable to use the hypoglossal, in my opinion, at the first operation; possibly not at all, as the small branches have been cut instead of the larger ones. I think he can be at least made comfortable, and better chewing facilities can be obtained by this operation."

He was referred further to Dr. Dean Lewis for opinion as to the possibility of repair of the nerve injury, and Doctor Lewis agreed with Doctor Potts that the injury had been done at the time of the operation, to the smaller branches or after the seventh nerve divides, and, therefore, it would be practically impossible to reunite them with any hope of success, and so far as the nerve was concerned, the operation would not be advisable.

*Comments.*—There were two mistakes made in this case, apparently. The first, the fact that no serious attempt was made to hold the fracture in apposition by wiring the teeth together, supplemented by a cup splint under the lower jaw, or a simple snug bandage around the head, assisting the wires in holding the teeth in coaptation. The second, the fact that the operation was done without apparently proper knowledge of the anatomy of the parts to be traversed between the surface and the object of the operation, namely, the lower maxilla. The reason for the displacement in these cases of fracture of the neck of the condyle is that the upper fragment is drawn upward and forward by the external pterygoid, the remainder of the jaw is drawn upward by the masseter temporal and internal pterygoid. This deviation sometimes leads to excess callus and interferes with mastication, following this injury, and if it was necessary to operate following proper efforts to maintain the fragments in apposition, then, if the surgeon was not familiar with the anatomy of the parts involved, he should have familiarized himself before attempting to do the operation.

The seventh nerve, which supplies practically all the facial muscles of expression, exits from the foramen in the mastoid, passes through the parotid gland, dividing about the anterior margin of the ear and spreading over the entire side of the face. It is entirely a motor nerve at its exit and supplies none of the sensation to the face, but all of the muscles of expression. In this case a vertical incision 1 inch in front of the ear, cut directly across the upper half of this nerve which supplies all the muscles above the junction of the lower

and middle third of the face. Had it been cut in its main trunk, it could have been sutured with some hope of success, but inasmuch as it was cut in the finer fibres, of which there are many (see Fig. 22), it would be a superhuman task to attempt any repair.

This man is condemned to wear an eye-shield for the rest of his life to prevent particles of dirt and dust from striking the cornea and producing corneal ulcers. He will be constantly annoyed by the overflow of lachrymal fluid from the lower lid and the eye will not be bathed normally, so that it will give him an almost constant dry sensation of the eyeball. This would seem to be a terrible responsibility chalked up against any surgeon, because it certainly could not have been a surgical accident, inasmuch as the original incision was not planned to avoid this nerve, but was planned so that it cut immediately across the fibres, taking into consideration only easy access to the fracture and not the importance of the location of anatomical structures or of cosmetic result. It, therefore, was ignorance and conscienceless operating and not a surgical accident, which may happen to the most conscientious operators.

#### SUBDELTOID BURSITIS AND PARTIAL DISLOCATION OF THE ACROMIO-CLAVICULAR JOINT

C. W. B., age fifty-two, carpenter.

*History of Present Condition.*—In February, 1920, this man slipped on some ice and fell, striking the point of his shoulder against an upright. He had immediate sharp pain which was referred to the whole shoulder area covered by the deltoid, was unable to continue work on account of the pain. There were very slight external evidences of injury, according to his own statement. His arm was placed at his side and held in place by adhesive plaster straps around his body, for the purpose of rest. It was removed in about ten days from this dressing, and he carried it in a sling for some two weeks longer, after which time he was told to use it. This he tried to do, and as a matter of fact, did, in the antero-posterior motions and abduction as far as about 45 degrees. Beyond this, he was unable to raise his arm. He was given massage, but this seemed to irritate the condition and the shoulder gradually grew worse until he was unable to sleep a good part of each night on account of the pain in the shoulder and a hot water bag was his constant companion. He had especially severe pain when he turned on the affected side, which threw weight on the shoulder. During all this time the massage was continued. It was finally decided that the patient did not want to work and recommendation was made to discontinue his compensation.

*Examination* (May 22, 1920).—At this time, some three months after the injury, patient appeared for examination, complaining of the

symptoms just described. Inability to abduct the arm and pain in the shoulder, the pain being referred to the whole deltoid area. Upon examination it was found that the antero-posterior motion was normal, that external rotation was painful and even, passively, it was impossible to externally rotate the arm beyond the antero-posterior plane. There was tenderness over the acromioclavicular joint and some tenderness over the greater tuberosity. Voluntary motion was limited to swinging the arm forward and backward at the side through an arc of about ninety degrees. Abduction was impossible, actively or passively, beyond forty-five degrees, and it was found that most of this motion was between the scapula and the ribs rather than in the shoulder itself. When the patient's arm was held at the side pressure made directly upward on the elbow, pain was elicited. Patient was unable to put his hand in back of him in complete internal rotation. Pressure made at the point of the shoulder in a line with the axis of the clavicle, gave severe pain at the acromioclavicular joint, and it was noted that the distal end of the clavicle on the affected side was somewhat more prominent than on the opposite side.

A diagnosis was made of partial dislocation of the acromioclavicular joint, which was probably the primary injury, complicated by a subdeltoid bursitis, which was the result of fixation of the arm at the side for a considerable period of time. It was believed that there was also some inflammatory condition in the acromioclavicular joint as the result of over-manipulation of the joint, the ligaments of which had been injured and the cartilage had been damaged by rough treatment.

Operation was advised for two reasons: First, to fix the acromioclavicular joint by suturing and, incidentally, to examine the surfaces of this joint to see whether there was any inflammation present and if so, to resect the joint; and second, to treat the subdeltoid bursitis by the Sir Robert Jones method. The joint was found to be inflamed and the acromioclavicular ligaments torn. This was sutured in the manner described in the last issue of the CLINICS.

*Sir Robert Jones Manipulation for Subdeltoid Bursitis.*—While still under the anæsthetic the Sir Robert Jones method of manipulation was instituted, which consists of bringing the patient's arm up to right angle with an assistant's hand holding the external border of

the scapula firmly against the ribs, so that the motion is confined to the shoulder. Also have an assistant's fist in the axilla, pressing against the head of the humerus to prevent any dislocation downward of this joint. The arm is moved forward and back and gradually abducted until a right angle is reached. After this has been accomplished, the arm is quickly and forcibly brought into hyperextension; that is, the elbow being brought back to the full normal range. This is calculated to tear apart the surfaces of the bursæ and allow normal range of motion subsequently. The arm is then put through its full range of motion. It is sometimes extremely difficult to obtain the ninety degrees of abduction, and care must be taken to see that the scapula is immobilized and pressure is put under the head of the humerus or a dislocation may easily result. Subsequent treatment consists in holding the arm in complete abduction and external rotation, and instituting motion for at least ten minutes, three times a day, for a period of two weeks, especially complete abduction and anterior and posterior motion with rotation while in the abducted position.

#### SHOULDER INJURIES

There is probably no other joint which is so frequently injured and gives such consistent trouble as the shoulder. An effort has been made to boil down certain facts regarding painful shoulders in an attempt to make them more or less understandable and bring attention to the things which are already known, but which probably, in the stress of other demands have not been put into concrete form in regard to shoulder injuries.

A full understanding of the anatomy of the shoulder-joint, especially that of the bones, capsule and muscle attachments, is necessary for an understanding of the disabilities which so frequently occur from comparatively slight injuries to this joint. In the first place, we have many interlacing muscles which control a very lax joint capsule of the shoulder-joint, being so loose that it plays no part in the retention of the head of the humerus in the glenoid fossa except on extremes of normal motion, the normal tone of the muscles being entirely responsible for the approximation of the joint surfaces.

The muscles may be roughly divided into three groups: posterior, anterior, and external group. The external or the supraspinatus and deltoid act as abductors of the arm with the infraspinatus and

teres minor assisting in extreme abduction, but being ordinarily external rotators. These constitute a comparatively weak group of muscles. When it is considered that the anterior muscle, pectoralis major, and the posterior group, the latissimus dorsi, subscapular and teres major, which are as strong in adduction as they are in internal rotation, it is easy to see that the two weak motions of the arm are abduction and external rotation. The long head of the biceps, of course, helps somewhat in abduction, and the long head of the triceps quite counterbalances this in adduction. Add to this the assistance of gravity and we find that the abductors of the arm have, on an average, much poorer leverage and are much weaker than the adductors.

We have, further complicating this system of muscles, several bursæ which are subject to all the ills of endothelial-lined sacks, to say nothing about the tendinous attachments of the muscles. The subcoracoid, subdeltoid, and subacromial bursæ and the tendon sheath of the long head of the biceps are all extracapsular, although they can be considered as part of the joint, since any disability in any of them limits somewhat the motion of the joint. Given an injury which traumatizes the shoulder-joint or its attached muscles, it is impossible to miss one or more of these bursæ (Fig. 23).

We have all been taught from the days of our undergraduate work until very recently that the proper dressing of shoulder injuries for immobilization was a Sayre's or Velpeau dressing; in other words, complete adduction with internal rotation, the hand resting on the chest and the forearm and arm firmly tied to the side and front of the chest. In view of the fact that in this position the deltoid is pulled tight, the supraspinatus is stretched, the infraspinatus and teres minor are put on tension and the adductors and internal rotators entirely relaxed, there are several things which occur which it will be hard to correct following the mobilization of this joint.

When the deltoid and supraspinatus are pulled tight, the head of the humerus is forced up snugly against the acromion process, putting pressure on the subacromial bursa, which is often continuous with the subdeltoid bursa. The surfaces of the subdeltoid bursæ are pressed tightly together between the greater tuberosity and the acromion, and the long head of the biceps is stretched tightly over the curve in the bicipital groove. The weak group of muscles, namely,

the deltoid, supraspinatus, infraspinatus, and teres minor are pulled tight, and the stronger muscles which have the greater leverage, namely, the pectoralis major, latissimus dorsi, teres major and subscapular, are relaxed and allowed to contract.

We are all familiar with the facility with which endothelial surfaces become adherent when pressed tightly together in immobilization. We are also all versed in the rapidity with which muscles become contracted more or less permanently when allowed to be disused and where the tension is removed. The normal tone of the muscle seeking a certain resistance and contracting until it meets this resistance. Now, when the arm is removed from its immobilization dressing, we not only have to overcome the contracture of the adductors and internal rotators, the stronger group, with an overstretched, weaker group, the abductors, but we have to overcome the weight of the arm or the pull of gravity assisting this stronger group, and we have to break up the adhesions which have formed between the endothelial surfaces of the numerous bursæ just mentioned. This we try to do by telling the patient to use the arm, or we may aid him by massage a half hour a day or a half hour every two days. The rest of the time the patient allows the arm to rest at the side, because this is the most comfortable position for him, or he makes feeble efforts to use it, the efforts being necessarily feeble because the groups which make the effort are the weaker groups. He can move his arm forward and backward without pain because this involves nothing except a perpendicular swing and the motion of the head of the humerus against the glenoid fossa, and he complains bitterly that he is unable to get his arm away from his side more than thirty degrees, which motion, if carefully analyzed, is found to be between the scapula and the chest wall and not between the humerus and the glenoid cavity.

Upon examination it will be found that the external rotation beyond the antero-posterior plane will be impossible without severe pain. Abduction will be impossible beyond forty-five degrees on passive motion, at which time he will complain of pain at the end of the acromion and over the greater tuberosity. It will be noted that the adductors and internal rotators are tense, the anterior axillary fold will stand out prominently, and it will be impossible for the patient to relax the muscles constituting this fold. There will be a tender spot about the size of a quarter to a half dollar over the greater



tuberosity, and there may be a smaller spot over the lesser tuberosity, which is also tender. Pressure upward on the elbow with the arm at the side, throwing the patient's weight on the shoulder, pulling the deltoid tight, will give pain, as will extreme internal rotation and abduction; that is, flexing the forearm and trying to put the hand in back of the body, because this draws the supraspinatus and deltoid into extreme tension and forces the head of the humerus and greater tuberosity up against the acromion and the deltoid. This, of course, is a condition with which we are all familiar, namely, subdeltoid bursitis. The X-ray in the large majority of cases will show a normal joint. In some few cases, especially in those of patients of past middle life, there may be seen at the tip of the greater tuberosity a faint shadow as of deposit of lime salts in the bursæ. It will depend upon the angle at which the X-ray is taken as to whether this calcification of the subdeltoid bursæ will be apparent. The arm must be externally rotated so that the greater tuberosity will be brought exactly in profile if the rays are thrown straight down; otherwise, the shadow of the greater tuberosity and the calcification of the subdeltoid bursæ will overlap. Granting that these things are true, it would seem that the methods which we have been taught to pursue in the treatment of shoulder injuries would lend themselves to producing a permanent disability rather than producing a cure of these injuries. In fact, that is exactly what occurs in the large majority of cases. A comparatively slight injury to the shoulder put up in the usual way with the arm at the side and the hand and forearm on the chest, will produce a more serious disability in a considerable number of cases than if the original injury had not been treated at all.

We must, then, change our entire method of procedure when dealing with shoulders, namely, instead of being fixed in adduction and internal rotation, they must be fixed in complete ninety-degree abduction and external rotation, not only to allow the weaker group of muscles to go into contracture, if necessary, or at least into relaxation, and the stronger ones to be put on the stretch, but to take the pressure from the subdeltoid bursæ by relaxing the deltoid and supraspinatus muscles, which automatically allows the subdeltoid bursæ room. The external rotation throws the greater tuberosity back of the acromion process; the head of the humerus down in the joint, away from the acromion; and pulls the lesser tuberosity backwards, away

from the coracoid, at the same time relaxing the long head of the biceps. This is a somewhat awkward position, it is true, but until we realize that it is necessary to do this to avoid permanent disabilities of the shoulder from slight injuries, we will continue to have patients who go about unable to do their normal work because the treatment was worse than the disease.

To put the arm up in a cast in this position is extremely difficult, to say nothing about being extremely uncomfortable, because the weight of the arm constantly presses down on the side of the chest wall, which takes the weight and the edge of the cast jams into the patient's side and produces a very uncomfortable situation. There is, however, a method of dressing in ambulatory cases which is quite comfortable and is easily applied, namely, a modification of the aeroplane splint, which we call the Cleary splint because it was designed by Captain Cleary while at the Lakewood, New Jersey, Hospital during the recent war (see illustration Fig. 24). It will be seen from the illustration that the arm pressing down, throws the weight entirely on the belt and the opposite shoulder, these two points taking the leverage, the opposite shoulder being the fulcrum of the lever, the injured arm and the hip on the same side, being the two ends of the lever. These points are pressed upon, not by hard plaster, but by soft leather which moulds comfortably to the body over the well-padded bones and the patient may be allowed to walk around and be comfortable while at the same time maintaining the arm in the proper position.

If the case is not ambulatory, this position is easily maintained by tying the hand of the injured side to the head of the bed. This maintained full external rotation and complete abduction of the arm. If it is desired to put extension on in the case of fractures, it is easily done by a well-padded traction around the elbow-joint, and it is our opinion that every injury to the shoulder-joint, regardless of its seriousness, should be treated, at least for a time, in this position. If partial fixation takes place in this position, when the patient is allowed to become upright again or to remove the splint, the force of gravity will gradually break up any adhesions which have formed, especially when they are aided by the strong adductor muscles, and if massage is established, the full normal range of motion is regained in a very few weeks; whereas, if the treatment is in complete fix-

tion with the arm at the side, it takes many months to establish a cure, and sometimes a cure is impossible to establish.

There are other things which may be taken into consideration in connection with every shoulder injury, the principal one being chronic infections. We are all very familiar in these days with the possibility of chronic infections, which are unobserved until some traumatism causes an inflammatory process in a joint, ligament, tendon, or muscle, which will not clear up under ordinary methods of treatment, and refuses to be relieved until such time as these infections are cleared up. Many of our shoulder-joint injuries have superimposed upon them the results of infections or toxæmias which keep them in a constant state of irritation and giving constant pain. It is hardly necessary to point out here that in case of injury to any bone, joint, tendon or ligament, it should be the invariable rule at the time of the first examination to give the patient a thorough overhauling to determine whether there are any infections of the teeth, tonsils, accessory sinuses, gall-bladder, appendix, prostate and seminal vesicles or urethra, or whether there is any indication of intestinal toxæmia, which will have a tendency to draw out a traumatic inflammation which should clear up promptly under ordinary methods of treatment.

In fractures of the anatomical and surgical neck, it is our opinion that both these injuries should be fixed operatively within a few days after their occurrence and the arm placed in the position of complete abduction and external rotation, after such fixation. The fixation should be firm enough to allow of passive motion within two weeks of the injury. In the author's hands, the driving of an ivory screw through the shaft of the humerus into the head has given most excellent results. This is an easy method of firm fixation, and, because there is little or no leverage applied by the muscles attached above the surgical neck, it is possible to institute passive motion very early in these cases.

In dislocations of the head of the humerus we find in a considerable number of cases that in the passage of the head forward and downward, attachments of the muscles to the greater tuberosity have torn a shell of bone from this tuberosity and displaced it backwards (see Fig. 25), pulling it up against the internal surface of the spine of the scapula where it comes over to make up the acromion process, and this small shell of bone is impossible to replace through the ordi-

**FIG. 25.**



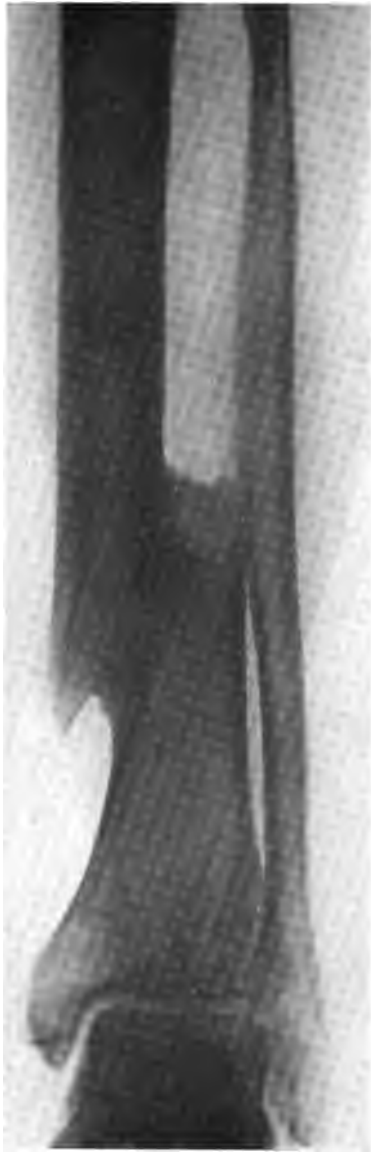
**Fracture of greater tuberosity following sub-glenoid dislocation of shoulder. This fragment is not displaced as in some cases, which are pulled backward and upward by the supra and infra spinatus muscles.**

**FIG. 26.**



**Mechanism of dislocation of outer end of clavicle. (G. G. Davis, Applied Anatomy.)**

**FIG. 27.**



**Back pain due to faulty balance. Antero-posterior view showing fracture of the right tibia with displacing of the upper end of the lower fragment towards the fibula, overlapping the interosseous space.**



**FIG. 28.**



**Back pain due to faulty balance. Lateral view showing fracture of the right tibia at junction of the lower and middle thirds with the overlapping that caused shortening of one inch.**



nary incision for shoulder operation. It is necessary here to make an incision, as pointed out by Phemister, along the spine of the scapula, through the posterior part of the deltoid muscle, pulling the infraspinatus and deltoid downwards and backwards. The fragment can then be grasped and by external rotation and abduction of the humerus, the greater tuberosity can be brought up into position to receive the shell of bone which has been torn off. This may be sutured, preferably with some absorbable material, to the shaft. It will be found that it is not of sufficient thickness to hold any other than a suture form of material. It is a somewhat difficult surgical procedure to replace this shell of bone, but in order to reestablish normal shoulder motions, it is necessary to see that this fragment is properly and promptly replaced, and in every subglenoid dislocation of the humerus it should be looked for in the X-ray, and if found, should be replaced immediately.

Complete or partial dislocation of the outer end of the clavicle is not an uncommon injury and is entirely overlooked, especially when not complete. A blow on the acromion process or tip of the shoulder, either from being thrown from a moving vehicle, landing on the point of the shoulder or having something drop, striking a heavy blow on the point of the shoulder, brings into play a leverage which tears the acromioclavicular ligaments, and in some cases where the force is continued downward, the coracoclavicular ligaments. The coracoid lays at about the junction of the outer and middle third of the clavicle, immediately under it, and it will be seen that a blow on the acromion carries this bone with the clavicle downward and outward, rotating it on its antero-posterior axis. This throws the acromion down and the coracoid up, the coracoid acting as the fulcrum with the two ends of the lever at the two ends of the clavicle. If this force is continued on farther, the scapula is forced farther down and the coracoid pulled outward and downward so that the coracoclavicular ligaments are torn, and then the sternomastoid pulls the clavicle straight up at its inner end (Fig. 26). This dislocation is easily replaced, but is extremely difficult to hold in reduction, since there is nothing on which one can apply a leverage, and in these cases it is invariably the rule that a suture of this joint is necessary.

This joint is also subject to all the ills to which other joints are subject and may become inflamed through trauma or when partially



dislocated. Where there is only a tearing of the supports of the acromioclavicular joint, it often is painful with the pain referred more particularly to the deltoid muscle and its attachment. When the acromioclavicular ligaments are torn it is apparent that the acromion will drop slightly outward and downward and throw a strain on the fibres which are left, which gives a painful shoulder with very few other symptoms, except localized tenderness over this joint. In cases of this kind a suture to support the joint will relieve it. In chronic inflammatory conditions of this joint, a resection of the joint with some sort of suture following, gives immediate and permanent relief, and is an operation which has not been given enough consideration.

Habitual dislocations of the shoulder should be mentioned in passing. The operations for the correction of this condition are familiar. These are curable in a large majority of cases by plication of the capsule as described by T. Turner Thomas, or excision of that portion of the anterior inferior aspect of the capsule and narrowing it by overlapping the edges, or deltoid flap operation.

In conclusion, it is to be emphasized that all shoulder injuries should be treated by ninety-degree abduction and external rotation, that early passive and active motion be established and, if possible, dry heat and massage applied at the earliest possible moment.

#### BACK PAIN DUE TO FAULTY BALANCE

J. H., age seventy, miner.

*History of Present Condition.*—On May 28, 1918, this man was caught in a fall of slate, while working in a mine, sustaining a fracture of the right leg at the junction of the lower and middle third. He claims now that he has pain in his right leg, radiating up into the back and that he is unable to work on account of pain in his back.

*Examination* (August 12, 1920).—Examination of the right leg shows a sharp protuberance about  $1\frac{1}{2}$  inches above the external malleolus, very good weight-bearing line, slight deviation of the lower fragment towards the outside. Right leg measures from the anterior superior spine to the internal malleolus  $28\frac{1}{2}$  inches; left leg between the same two points measures  $29\frac{1}{2}$  inches (Figs. 27 and 28). There is no swelling in either leg. Middle of the right thigh, circumference is 14 inches, left,  $13\frac{3}{8}$  inches. Movement of the hip, knee and ankle on the injured side is normal. The patient, stripped, stands listed a

little to the right. He is able to stand on the right leg and flex the left thigh against the body, and is able to do the same with the right leg when standing on the left. Lateral motions of the spine are normal. Forward motions are normal. Knee jerks are normal and equal on both sides.

The examination and X-ray show good solid bony union of the fracture with a sharp spicule of bone protruding from the lower end of the upper fragment downward and inward, with some overlapping of the interosseous space, the lower fragment being displaced outward, but the line of the leg being good.

The blood-pressure is 175 systolic, 65 diastolic. The heart sounds are somewhat weak, the area of the heart larger than normal.

*X-ray pictures* taken by Dr. H. E. Potter show, besides the fracture, hard, pipe-stem arteries, these arteries showing in the rays of the hip, spine, and chest as well as in the leg.

*Comments.*—The thing that this man complains of now is pain in the back, and yet he complains of no injury to the back. It is plainly evident, after examination, to what this pain is referable. Here is a man of seventy years, with hard arteries, poor circulation, and rigid ligaments, who is thrown out of balance by shortening in one leg, which throws the strain on one group of muscles and ligaments in the back, causing them to hold more than their due share. In other words, he stands like the leaning tower of Pisa, without a brace except the muscles on the side opposite the short leg. This amount of shortening in a child could easily cause a marked scoliosis by cross strain on the ligaments and muscles, but in a man of this age the bones are hard and ligaments are rigid, there is no chance for the ligaments and bones to mould, and consequently the strain is thrown on the muscles; and constant overstrain of any group of muscles or ligaments gives constant pain.

*Treatment.*—The shoe on the affected side should be built up enough to compensate for the loss in length of this leg, thereby re-establishing the man's normal balance and relieving his back muscles of the excess strain of carrying excess weight.

# Pædiatrics

EDITED BY JOHN FOOTE, M.D.

Washington, D. C.

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## EGG SENSITIZATION OF HIDDEN ORIGIN IN ECZEMA OF THE INFANT \*

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SOME years ago Von Pirquet used the word "allergy" to indicate the altered reaction produced in an organism when infectious material was reintroduced. While Fordyce in 1911 wrote of a probable anaphylactic origin of certain eczemas, it remained for Schloss in a later preliminary report of certain experiments to conclude therefrom that some conditions called eczema were due to "food allergy." Since that time the fact has been established that young children with eczema, when tested for a cutaneous reaction, give positive evidence that they have been sensitized by the protein constituents of one or more substances used in food.

These tests, as you know, consist of (1) either injecting a weak solution of properly prepared protein between the layers of the skin—the *intradermal method*—or (2) gently rubbing a slightly abraded or cut portion of the skin with some of the protein dissolved in decinormal solution of sodium hydroxide—the *cutaneous method*. The latter procedure is more reliable and safer than that of the intradermal procedure—safer, because in cases of extreme sensitization the intradermal injection may occasionally, even in practiced hands, produce a disagreeable general reaction. More than one commercial preparation of proteins for these tests are now on the market and are fairly reliable.

The investigations of various observers have shown that egg white or some constituent of egg is frequently the cause of skin irri-

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\* A clinical lecture delivered before the third-year class of the Georgetown University Medical School, Washington, D. C.

tations, and it is almost a routine procedure to make tests for protein sensitization in children who have eggs in their dietary. However, I wish to present two examples of children who give a positively negative history of feeding with eggs—and yet, in the face of this, show decided reactions when tested for allergy to egg protein.

R. A., aged two and one-half years. Has had eczema since thirteen months. Weeping and crusted type spreading over the face and the chest and the hands. Has persisted in spite of all sorts of restricted diets. Is a well-nourished boy, face much swollen, eyes almost closed. Family history shows that neither mother or father had eczema since childhood.

Present dietary is milk, cream of wheat, and graham wafers.

Test for wheat—no reaction. Control, negative.

Test for milk—no reaction. Control, negative.

Test for egg—both white and yolk show after ten minutes an area of white skin, slightly stellate and raised, 1 cm. in diameter, surrounded by a reddened area.

In performing this test a small dental drill is used after the manner of a Von Pirquet scarifier to remove only the outer layer of the skin. The control is made a short distance away. A small portion of dried egg protein is rubbed into the abraded surface and a drop of decinormal sodium hydrate solution is rubbed in with a toothpick in order to dissolve the protein. You will notice now in a few minutes a white, raised surface, surrounded by a reddened area. This white surface ought to be at least three times the area of the control abrasion, which is perfectly round, and also slightly blanched.

The reason for the reaction in this child is found in the fact that, in spite of his parents' denials, he was really receiving egg albumin in the graham wafers fed to him. Many sweetened preparations for children, graham crackers, sweet crackers, sweet zwieback, etc., contain some egg. It is necessary, therefore, to forbid these articles in the dietary of such children. In the present instance, forbidding these foods caused an immediate cessation of acute symptoms.

The next case is that of a child three years old. At fifteen months he showed an eczematous eruption covering the entire body. As he had been given egg every day, his mother was advised to omit this article from his diet. He not only improved, but a symptomatic cure occurred. Last month he was brought to me with a very acute eczem-

atous eruption on his forehead and face. He had not been given any egg, but his mother remembered that a week before the appearance of the eruption he had wandered into the hen-house and broke an egg over his head and face. The eruption appeared within two or three days—whether by contact or from swallowing some of the egg is not clear. A report a week later showed rapid improvement.

Another instance of sensitization to egg was found in an infant that contracted eczema at three months of age, after breast feeding had been discontinued. No history of eczema occurring before this time could be obtained. The attack was an acute type with involvement of the face, scalp, and portions of the body and extremities. The infant was given proper and usual treatment by the attending physician, and later was seen by two dermatologists. Little relief was obtained. The little patient was eight months old when I saw her. She had been slowly improving for two months when suddenly a most violent recurrence came about. After inquiring as to the diet, routine tests for various proteids were given. Milk, rice, oat-meal, and stale bread were alleged to be the only foods given. From the history it might be assumed that the employment of cows' milk in the dietary could easily be the cause of a sensitization to milk proteid. The reaction to both cows' milk and human milk was negative. Wheat also gave a negative reaction. But when whole egg protein was used a violent reaction was noted within a few minutes. A blanched area 2.5 centimetres in diameter appeared on the arm with a reddened areola surrounding it. The administration of egg in any form was denied. On inquiring whether any sweet cakes had been given, it was learned that the grandmother, who was visiting at the house, had fed "just a little" of some cookies, which she had presumably made herself, and so was certain they could not hurt the child. That this was not the cause of the original attack is quite certain, and it is quite probable that no protein sensitization reaction would have been found in this infant at first. This was probably an infant with the so-called "anaphylactic family history." That there are many such cases, and that children of this class are peculiarly susceptible to later sensitization to proteins, may explain the reason why the omission of the offending protein from the dietary of an eczematous child may not result in the complete symptomatic cure which might logically be expected. There are no doubt many such children in which protein

sensitization is facilitated by "the exudative" diathesis, and this sensitization does not initiate, but always serves to aggravate, the eczematous condition.

These examples show that the etiology of sensitization to proteids is sometimes difficult to locate at first. It may be laid down as an axiom, however, that a positive reaction is absolute proof, when properly controlled, of a food allergy, and any food, not elemental in type, which is being consumed at that time should be regarded as a potential cause, until proven innocent. Egg, especially powdered egg, is a frequently used constituent of sweet package biscuits and the like, and the fact that egg itself is not in the diet is not a sufficient precaution to effect relief in egg sensitizations.

## THE TEETH IN THEIR RELATION TO THE HEALTH OF THE CHILD

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DURING infancy and childhood there is a common tendency to relegate to the province of the physician matters relating to the eruption and diseases of the teeth. Indeed, it is unfortunately true that in many instances not even the aid of the physician is invoked in cases where the teeth are supposedly at fault. The lay mind is only too ready to attribute to teething a multitude of infantile disorders and to ignore as of trivial importance disease of these deciduous structures, because of the fact that the first teeth are destined to be ultimately lost.

In consequence, it is doubly important that the medical mind be alert to the responsibilities which the physician must assume when his advice is sought or when opportunity arises to impart the lessons of oral hygiene and of dental prophylaxis.

It is not to be denied that considerable evidence from a diagnostic standpoint may be obtained by careful routine examination of the teeth which further emphasizes the necessity for observing in detail the appearance of the dental structures in formulating conclusions concerning the general health of the child. Not infrequently evident neglect of the teeth, as revealed by even cursory examination, will direct attention to gross oversight in other matters of the child's personal hygiene and may lead to corrective measures of great importance.

It is passing strange that structures of so vital importance as the teeth, the guardian outposts of healthy alimentation upon which life itself depends, should be so often neglected in the salvage of child health.

In the animal kingdom the prehensile function of the teeth exceeds that of the forefeet and claws. The human who, in later years at least, lavishes the utmost care upon his digits, forgets the value which comparative physiology assigns to the dental organs.

There can be no doubt that in the earlier centuries the very quality of food obtainable with the necessity for better mastication and conse-

quent functional activity, created conditions more favorable to the preservation of the teeth. It is well known that the teeth of the Egyptians which have been subjected to modern scrutiny through the study of mummies, not excepting those of the Egyptian children, were far better preserved than the teeth of the present-day individual, either child or adult.

The refinements of food preparation and cooking, the use of so many soft foods, all tend to promote tooth deterioration both by belittling their normal function and by permitting clinging deposits to remain upon and about the dental structures.

#### THE SCAPEGOAT OF INFANT ILLNESS

The apathy of even the modern mother in assigning to the eruption of the teeth every possible symptom in the category of disease is appalling. This conception is too often shared by the practitioner who may be rudely awakened from his false belief by the development of an otitis media, or even mastoiditis, pyelitis, or what not, which have been masked in the shadow of an erupting tooth. Even the mother herself is not so sure—witness the discrepancy in the topographical location of “eye” teeth and “stomach” teeth, terms which are indiscriminately applied to lateral incisors, canines and first molars, in a jargon of legendary inaccuracy. Witness, also, the quite too common belief on the part of the mother that the sixth-year molar belongs not to the permanent teeth but to the deciduous or milk teeth. Should decay threaten the loss of this tooth, probably the most important unit in the youthful jaw, conservative treatment is often unsought because of the erroneous belief just noted.

#### THE NEGLECTED PRE-SCHOOL AGE

Dental literature is replete with writings relating to the neglect of the teeth of childhood, yet but few records are found in medical literature in emphasis of this important part of health conservation.

Much attention has of late been directed to the pre-school child, the individual who has passed beyond the infant care and supervision now extensively accorded him, but has as yet failed to enter the domain of school inspection, including, as it does, dental prophylaxis. This period, from two to six, has heretofore been largely the unobserved epoch of child life so far as pertains to periodic examination of the



supposedly healthy child. Yet it is this especial period in juvenile growth which witnesses the greatest ravages of the acute contagious diseases, which is prone to contain the largest number of those suffering from malnutrition and undernourishment, and which shows the most flagrant disregard of proper and balanced dietaries.

Not only are the "defects" of the pre-school age those which arise during this epoch of life, but they are not infrequently—and particularly this can be said of the teeth—legacies of the first and second year, even of the pre-natal period itself.

#### THE MATRIX OF THE PERMANENT TEETH

The permanent teeth, unfortunately, lie veiled and hidden from view for years prior to eruption, nor is there a proper visualization that their delicate foundations are well under way even before birth. Indeed, of each and every tooth possessed by man might be said in the words of Longfellow:

For thee was a house built ere thou wast born,  
For thee was a mould made ere thou of mother camest.

For thus is the superstructure elaborated before the birth of the child, and only too often, notably in the case of syphilis, the legacy of deterioration and death has been stamped upon the dental organ even before individual existence began.

The old adage, "a tooth for every child," was resultant upon the time-honored neglect of the pregnant mother. This has fortunately given place to rigid dental prophylaxis and with other equally important measures of maternal supervision coming into vogue much will be accomplished toward securing for the offspring a healthy birth-right, with its accompanying corollary, a healthy dental equipment.

Maternal nursing is equally as responsible for normal, healthy teeth as for healthy bodies. The delayed and imperfect dentition of the artificially-fed is never questioned.

#### THE CRIMINALS IN THE DRAMA OF INFANCY

The criminals of the baby's first year are rickets, scurvy and malnutrition, all these products of bottle feeding and more especially of proprietary feeding, which sap the vital structures of the teeth with the other structures of the body.

It is of common observance that the teeth are very late in appearing in infants suffering from rickets and that their successive eruption, at times, may be associated with rather marked constitutional disturbances to which the rickety infant is especially prone because of his spasmophilic diathesis and marked leaning toward latent and demonstrable tetany. While at times the teeth after eruption appear to possess normal vigor, such is not usually the case, for, sooner or later, they seem destined for the ravages of early decay and deterioration. This is especially true of the incisors, which even at a tender age show erosion and serration, saw-like markings of the cutting edges and atrophy of the neck or alveolar end of the tooth, changes which are sometimes simulated by syphilitic teeth, but which, in our experience, are much more manifest in rickety infants.

Not only the constitutional dyscrasia of rickets, but that of thyroid insufficiency, results in markedly delayed dentition.

The taint of scurvy affects especially the upper gums and after the eruption of teeth one may witness boggy infiltration, hemorrhage and even ulceration about the upper incisors. Fortunately, the detection of the disease, with consequent abatement of symptoms under rational dieting usually restores the dental organs to normal. From the standpoint of diagnosis, the purplish, swollen, overlapping gingival margins about the upper incisors are of the greatest moment. Even in the absence of erupted teeth this specific gingivitis will be found in many instances.

#### HEREDITARY LUES AND THE TEETH

The deciduous teeth rarely show characteristic changes due to inherited syphilis. There may be early erosion, decay and serration, but the manifest stamp of lues must apparently be exerted at that stage of tooth development when dentinification is taking place within the alveolar nests. This period, relating to the temporary teeth, occurs early in pregnancy, so that syphilitic poison during this period of fetation, of sufficient virulence to leave tell-tale evidences upon the milk teeth, usually results in early miscarriage.

The permanent teeth in which dentinification occurs shortly before and for several months after birth may appear as the notched, peg-like incisors of Hutchinson, and the less mentioned, but equally charac-

teristic sixth-year molars with their irregular cuspal erosions of congenital lues.

#### THE EFFECT OF MECHANICAL AND FOOD INJURIES

Leaving aside the stigmata of somatic disease, there remain certain lesions which appear more or less local manifestations of tooth pathology but in which the rôle played by constitutional states cannot be entirely eliminated, except in the case of trauma.

Traumatism is not at all infrequent when one considers the age of the little patients and their greater liability to injury from falls and accidental blows. Deciduous teeth are commonly entirely dislocated from their sockets or broken in irregular ways—thus opening the avenues of the deeper structures to infection.

From the very nature of the foods during the early years of life, dental decay is actually encouraged. The larger part of the diet is with intent administered soft and finely divided. It consists largely of carbohydrates and not a little of sugar. Such residues remaining about the dental structures form favorable culture material for the acid-forming organisms which in turn attack the tooth substance.

Once initiated, the carious processes proceed with such rapidity that not infrequently a mere shell of tooth remains, filled with a vascular protuberant granuloma, often exquisitely painful.

It would seem needless to emphasize the secondary effects of such degenerations, yet it is the disregard of such possibilities which renders their development favorable. Stomatitis, gingivitis, pericemental and alveolar abscess, actual necrosis and loss of substance of the maxillæ, may take place in train. Adenitis of the adjacent cervical glands is a necessary corollary, nor can it be questioned that such adenitis, initiated by a septic focus in a tooth, may later succumb to tuberculous invasion.

Such severe local extensions usually lead to corrective measures but a host of by-products, so to speak, often more deleterious in their constitutional bearing are either overlooked or are tolerated as being of lesser importance.

#### ORAL FOCAL INFECTIONS OF CHILDHOOD

It is not far-fetched to believe that focal diseases in childhood may be equally responsible for continuous circulatory poison-

ing as are similar conditions in the adult. Rheumatism and joint disturbance, endocarditis and chorea, are easily within the range of such possibilities.

Morse, in a study of chorea, admits the likelihood of tooth infection in the etiology of this disease (*Am. Jr. Dis. of Ch.*, July, 1916).

Decayed, putrescent teeth, deciduous or permanent, favor the development of general oral sepsis inclusive of gums, buccal cavity, tonsils and pharynx. It has been demonstrated that the organism of ulcerative stomatitis, in reality a gingivitis, is identical with that of ulcero-membranous tonsillitis, as shown by Plaut and Vincent. It is largely held that the malignant noma of the mouth is due to the same infection which is so commonly found in the ulceration about the teeth.

"*Os sanum in corpore sano*" is to be desired even if many times wanting. Here at the atrium of the respiratory and digestive tracts may stand the vitiating Cerberus of a dying structure harboring a multitude of pathogenic organisms which need but a change of soil to take upon themselves renewed virulence.

The danger of mouth infection is recognized by the surgeon when he dons his operating mask; by the internist, when he masks his patients and himself to prevent cross-infection in the wards of a hospital.

*Streptococcus viridans* abounds in the putrescent pulp of a carious tooth. This organism, with its kindred strains, has of late been held responsible for an increasing number of dangerous conditions—notably in the lungs and pleuræ.

Thus the child with dental and oral sepsis becomes a health menace not only to himself but to his companions in play. To himself, because of the fact that medical science has yet to unravel the causes operating to activate and vivify the pneumococcus, the streptococcus and the like, which inhabit the mouth cavities of apparently healthy individuals.

By far the greatest number of cases of croupous pneumonia in childhood, as ascertained by grouping methods, fall into Group IV, which is the class found to inhabit many mouths in health, but what circumstance translates the apparently innocent oral guest into the virulent offender of disease is unknown. What should be patent is the evident necessity of making untenable the habitat of any pathogenic organism by as perfect oral asepsis as may be secured, nor

can this desideratum even be approached in the presence of carious teeth or diseased alveoli.

#### THE IMPORTANCE OF THE "FIRST" TEETH

The deciduous teeth are the splints upon which the orderly development of the permanent units depend for perfection of growth. The gradual absorption of their roots *in situ* as their governing function progressively ceases is no less the perfection of nature's orderly mechanism than the "gubernaculum" which guides the descent of the testis. The milk teeth are equally the gubernacula of their later successors.

Premature loss of the early teeth results in distorted development of the jaws, irregular spacing and manifest encroachment upon the positions allotted to the permanent group. It is, therefore, incumbent upon the physician, who often alone is cognizant of the condition of the child's teeth, to advise and secure adequate preservative care of the deciduous elements as a barrier against the evils of malformation and malocclusion which may ensue from parental apathy or ignorance.

During the first eight or nine years of life the temporary teeth almost alone function as the organs of mastication. Because of their evanescent existence this important fact should not be lost sight of, nor should their short life tenure be made the excuse for neglect in preservation.

It is obvious that a single painful tooth possessed by the child, from his voluntary effort to avoid pain, will put out of commission one-half of the coaptating jaws in the mastication of food. A single tender tooth on each side will utterly check all efforts toward mastication and lead to constant "bolting" of food.

It is unnecessary to dwell upon the train of digestive disorders consequent upon such improper functioning of the jaws. They include all degrees of anæmia, undernourishment, stunted growth, as well as the more frank disorders of digestion.

When it is considered that 25 per cent. of the school children in many centres of population show such abnormalities of growth, it is the obvious duty of the physician to direct attention to oral imperfections when they exist and to search for such possibilities whenever physical examination of the child is made.

## VIGILANCE THE PRICE OF COSMETIC RESULTS

Whether the growing child shall enter the adult state possessing symmetrical, coaptating and functioning dental arches, judged from both a cosmetic and physiological standpoint, rests largely upon the degree of watchfulness exercised by both parent and physician.

From the cosmetic point of view the debt one owes to the unknowing child to avoid and prevent facial blemish, especially of the girl child, is equally paramount with the necessity of preventing unsightly scarring in the surgery of childhood, vaccination wounds, abscess scars and like disfigurements.

It is equally necessary for the proper performance of the physiological function of the mouth, for the prevention of disorders of digestion, as well as for the very preservation of the teeth that normal coaptation of incisors and molars be secured.

During the early months and years certain clearly remedial conditions may obtain which later, if uncorrected, will cause mandibular distortion. Of these, obstinate thumb sucking, by constant leverage, pulls forward the upper alveolar arch while crowding back the lower—this during a period of development when the evil consequences of such an unchecked habit work irreparable harm. The older child may substitute pencil sucking to further accentuate this deformity.

Adenoid and tonsillar hypertrophy with consequent mouth breathing and palatal elevation give rise to a particular form of maxillary deformity, well recognized by the orthodontist.

A frequently encountered cause of the disfigurement of wide spacing of the upper central incisors is the persistence of the labial frenum or labio-gingival band which passes down and between these teeth. As this structure contains muscle fibre and is often quite stout and bulky, its early ablation by clipping, as commonly practiced with the lingual frenum, is to be advised. When but slightly developed, the labial frenum undergoes stretching and atrophy as the child grows older, but in young children, where its presence manifestly exercises a baneful effect upon the proper setting of the incisors, its removal is to be recommended.

The permanent teeth are tightly wedged together above the deciduous set. They furthermore outnumber their predecessors in the proportion of thirty-two to twenty. Any factor disturbing the contour

of the dental arch or militating against its normal, spreading growth, such as the early loss of the first teeth or the influences of co-existing evils just enumerated, produces distorted, irregular alignment or actual failure of eruption of the later teeth by permanent crowding out of important units.

#### THE PHYSICIAN SHOULD INSIST ON DENTAL TREATMENT

From what has been noted, it would appear reasonable to commend to the more thoughtful consideration of the medical attendant the possibilities of enlarging his diagnostic data by more careful inspection of the mouths of infants and children; to keep him mindful of the inherent dangers of oral sepsis in childhood as well as in adult life; and to emphasize his responsibility as often the sole dental and orthodontic adviser of childhood, in the discharge of which function he must insist on the early practice of dental prophylaxis and the employment of expert aid from his colleagues in dentistry when remedial defects are demonstrable.

Preservative methods applied to the temporary teeth should include the use of materials of such permanence that the structural integrity of the dental apparatus will make possible its maximum function until abrogated by the eruption of the more permanent organs.

The salvage of every possible temporary tooth to bulwark the spreading jaws in the orderly sequence of development should be more fully appreciated and more generally practiced.

#### ACHIEVEMENTS OF ORTHODONTIA

The correction of deformities of the teeth and jaws should begin at the very earliest period of child life, when the recognition of such conditions is made. Prior to the eruption of the "keystone" teeth, the sixth-year molars, much can be accomplished by way of anticipation of later deformities, including the abatement of pernicious influences, mouth breathing, thumb sucking, and the like, and even more by attention in detail given to the preservation of the deciduous teeth. Early banding of the sixth-year molars will make possible the exercise of the precision of orthodontic skill and will enhance the value of subsequent corrective measures as well as the rapidity of normal restoration.

Orthodontia has become a highly developed specialism of dentistry. Scientific principles of extreme nicety have yielded results which are not alone to be measured from their cosmetic accomplishments, a desideratum assuredly to be wished, but the perfection of the whole function of mastication must be accorded an even greater significance.

In conclusion, attention should be directed to the necessity of combatting the legendary myth concerning the horrors of dental work among children. Such ideas are usually the psychic reflection of magnified tales of fear and pain imparted to the juvenile mind from that of the adult.

No better argument has ever been adduced to prove the fallacy of this commonly accepted dictum than the experience of Doctor Ramsey, of St. Paul, among the children of France during the war.

He reports that the most popular member of his staff of child welfare workers was the dentist and he has brought back convincing photographs showing crowds of joyful, laughing children, actually clamoring for their turn to mount the dental chair! Theirs was not the psychic inheritance of the time-honored falsehoods of pain and suffering, for to these little repatriated refugees, and to their parents before them, dentistry was a luxury.



## PRECEPTS OF INFANTILE THERAPEUTICS

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THERE is no doubt in my mind that there exists a therapy belonging exclusively to children. There are anatomical, physiological and clinical reasons for this which do not require demonstration, showing the difference between adults and young subjects from the viewpoint of treatment of disease. In this paper it shall be my endeavor to show how infantile therapeutics should be understood, what should be its guiding principles and its method and in what way one should proceed in order to obtain—as often as is possible—the *cure* of the sick child.

Numerous are the *methods* which have been in vogue in general therapeutics; two of them—the pathogenic and the physiologic—are particularly in favor with the medical profession in France. But no matter how rational they may seem, they cannot be exclusively resorted to in the treatment of the diseases of childhood. Assuredly, to attack the known or unknown causes of disease, to attempt to dislodge or neutralize the morbid agent with all possible energy, is such a simple therapeutic and one with so sure an effect that no thinking person would oppose it. But regardless of the acquired results and the hopes raised, the circle of infectious diseases is not yet any too well understood, specific medication is a double-edged blade, dangerous to handle and, according to a celebrated remark, “*one may aim at the microbe and instead hit the patient.*” It must also be recalled that antiseptics, properly speaking, are, according to my limited experience, medicaments badly tolerated by children and that unless they be harmful in their action, they cannot be relied upon. As a single example I will take the failure of lactic acid that Hayem and others proposed as a sure microbicide in green diarrhœa. This drug acts simply as an astringent but is devoid of any other property.

Unquestionably, to isolate pathologic manifestations and to deal with them with therapeutic agents capable of producing contrary physiological effects is a method which is seductive on account of its

scientific aspect and from its promise of almost absolute certainty in results. It is the most profoundly scientific symptomatic therapeutics. But with Bouchard, it can be reproached on account of its disregard of the natural evolution of disease and for not taking the medical indications into account. It may also be dangerous when it attacks a phenomenon which is the result of a useful process; for example, a diarrhoea in a uræmic case or in some other form of toxæmia; a paroxysm of coughing in whooping-cough in a child with profuse secretions; a convulsive seizure in an hysteric, and lastly, fever in an exanthematic pyrexia.

But is it really necessary, given the actual state of our knowledge, to have a *method*, to profess a *dogma* in the treatment of disease? And does not wisdom consist rather more to select from everywhere the elements destined to form a final all—the judgment? Why should one deprive oneself of a procedure which is only bad when carried to exaggeration? Why not be *eclectic* and adopt such measures as may seem appropriate, according to circumstances?

There is nothing so deplorable in medicine and nothing that has done more harm to therapeutics than the spirit of system bringing about in turn infatuation and reaction, generators of scepticism. There is also a sort of mania among the members of our profession that should be guarded against, namely, to dose patients without rhyme nor reason with every conceivable drug on the market. Therapeutics is not an art to strike the centres of memory to gush forth a prescription, and it is especially in infantile practice that this deplorable tendency should be done away with. Take, for example, a child who has simply some fever, a little prostration and a coated tongue with mild gastric disturbance. Should quinine be exhibited, as is often the case in France, or, still worse, antipyrine, which, let it be said, is a very bad medicament for fever?

There is clearly no need of immediately attempting the reduction of the temperature, especially in children, because hyperthermia should not be confounded with pyrexia. There was a time when many lesions were supposed to result from a high temperature, such as muscular degeneration, degeneration of the heart and kidneys. Then fever was feared for itself and every means was resorted to to moderate it, but this practice has, fortunately, seen its day. Hyperthermy is not in itself a danger; it simply denounces a danger which menaces the organism,

likewise indicated by other symptoms which are present with the fever. Therefore, antithermic medication should not be employed, excepting when the rise of temperature, from its duration and from the symptoms which accompany it, points to some immediate danger; and the very best antithermic medication will be that which, while lowering the temperature, will most happily modify, in the most energetic and durable manner, the *ensemble* of the clinical signs manifesting themselves with the fever.

Let me add that fever is not infrequently a necessity, as, for example, at the onset of an eruptive fever or in certain intoxications; and finally let it be recalled with what ease—it might be said with what tranquillity—children sustain a high elevation of the temperature—when they are isolated—remaining seated in bed, a smile on their faces, as if they were not sick.

Expectation is consequently both an excellent and prudent practice at the onset of any illness in a child. The public, however, by its ignorance in matters medical, does not readily accept a physician who, when called to attend a child, simply folds his arms and awaits events. Therefore, without being true to one's convictions, other tactics can be resorted to. There is a drug whose sedative virtues are well known, and which, when judiciously handled in any case, is never dangerous; I refer to the tincture of aconite. It can be prescribed in order to gain time, because it is really a medicament of expectation. Very often, by its diaphoretic action and its sedative action on the circulatory system, it will cause the temperature to drop.

The family will often make much fuss if a strict diet is ordered, a practice common to many of our profession and very rightly so. To feed a patient afflicted by an acute affection, to fill the digestive tract with matter which will be incompletely elaborated by the altered gastric and intestinal juices, is simply to increase the probabilities of an intoxication. Fortunately, however, if the child is fed by friends against the physician's advice, three times out of four the stomach will instinctively rid itself of its dangerous overcharge.

It is also a difficult matter to keep a child in bed when it does not feel exhausted, and still it is only by rest in bed with proper supervision that a child can be properly observed. It is only by this means that the physician can interpret the various phenomena which arise, none of which is insignificant in children's pathology. In pediatric

practice there are no small details, the practice of medicine in children being made up of shades, and it is only by large experience with young children the physician can intervene usefully in their little morbid disturbances. Consequently, the bed, diet, a drink and aconite are the arms with which the announcing of the enemy should be dealt with, whose real virulence and nature is not yet known.

But supposing the case is one of severe bronchitis, typhoid or scarlet fever. Is it necessary to at once resort to the famous emetic which the older physicians always employed, but which is so badly supported by little children—especially the puny subjects who form the vast majority of hospital patients—as is, to say the least, useless, if not dangerous? In typhoid, should the child be at once subjected to balneation and his system filled with antipyretic products? Finally, in the case of scarlet fever, should serum treatment be applied when the eruption is in full evolution, the functional activity of the kidneys defective and simply because the laboratory report mentions the presence of the diphtheria bacillus in the mucus from the pharynx? No. Such practice, to my mind, is not in conformity with the data obtained by rational observation. It is not necessary to use a crowbar to kill a flea. For simple maladies, simple treatments.

The *naturist method* has rendered medicine much service by showing that disease has an evolution belonging to it, the result of the spontaneous effort on nature's part to counteract the infection. Therefore, it is, above all, necessary to favor this effort, either by imitating the natural processes or, when they are not understood, by furnishing the organism with those elements of defence of the highest possible energy. There are, in every disease, natural symptoms against which it is folly to struggle, so long as they do not transgress beyond the normal limits and do not rise to the rank of a complication. Nature alone prepares the cure and gives it its just measure; she toys with the artificial employed with the object of constraining her, and, according to Stahl's thought, she resists unceasingly, when it is a question of impressing irregular and inordinate movements upon her.

In the second place, it is most essential to endeavor to adapt one's action to the requisite effort and try not to prejudice the condition by hastily embarrassing the organism of the little patient with medicaments which are sometimes in themselves toxic or, on the other hand, by succoring him too long with violent medication. My hospital prac-

tice has demonstrated to me it is frequently a remedy badly applied, foolishly advised, that causes or keeps up the illness. I could quote many cases in point, but space does not permit. I know of two cases at least of children seriously ill with typhoid fever where it was an easy matter to become convinced that, after careful examination, the symptoms which seemed most alarming were simply due to a too-prolonged use of active drugs, such as quinine, antipyrine or carbolic acid, which when stopped caused the purely artificial peril to disappear as if by enchantment.

But now let us suppose that the disease is really existent with a train of serious phenomena which menace the existence of the little patient. It is time to intervene and strike quickly, forcibly and with exactitude. The progress of disease in childhood is frequently more rapid than in adults, and although young subjects recover more quickly, they are also more rapidly in danger on account of their lesser organic resistance and consequently die more rapidly than adults. What should be our conduct in these circumstances and upon what principles are we to rely?

The first thing to do is to make an exact diagnosis. To treat a case otherwise is deplorable therapy. Far better would it be to abstain from all therapeutic measures. But to recognize the disease, an already difficult problem to solve in children for those who are lacking in experience, is not, however, enough. It is essential to analyze "from top to bottom," so to say, both the patient and his case. Each organ, says Mauquat, in his excellent work, "*Traité Élémentaire de Thérapeutique*," each anatomic element may be the seat of reactions which impress their stamp upon the evolution of the disease in a given patient and impart characters to it as well which must not be overlooked by the medical man; without taking into consideration that the intensity of the causal factor may very probably also introduce an element in the production of the clinical variety. Consequently, it will be only after this minute analysis has been made that the clinician will determine his line of action. The nosological diagnosis may remain sometimes in default or remain uncertain, in which circumstance the functional state of the viscera may suffice as a guide. The essential is to perceive and seize upon the *indications*. I believe that all infantile therapy lies on this.

The *indications* are unquestionably complex and often multiple,

since they regard less the disease, its nature, form, intensity, malignancy or benignity, than the *personality* and state of strength of the patient, his morbid antecedents, the integrity or changes of the physiological functions. The indications also regard—at the time action must be taken—the order in which the series of measures shall be parallelly or successively employed which, usually, are combined or associated to compose what we call a medication.

On the other hand, I would insist on the fact that therapy is not a simple game of cards but a true mental operation relating to the choice of drugs and to the application to be made of them in given circumstances. This necessary subordination has been defined by Hayem: *the knowledge of the indications and the art of fulfilling them*. And as the indications are frequently multiple, the obligations of therapy should be likewise so. As bad as it is to prescribe several active drugs together, thus carrying out a polypharmacy whose least drawback is to prevent the physician from perceiving where he is going and where he will end, all the better is it to carry help to all the points menaced and to ward off the different necessities of the situation. In vulgar vernacular, “one must be everywhere at the same time.”

Suppose the practitioner has to contend with a severe case of broncho-pneumonia in rubeola. The lesions are not deep-seated but are disseminated. The child has a violent dyspnoea, a temperature of 104° F., excessive restlessness and a dry, incessant cough. The mouth and nose are the seat of impetigo. The abdomen is large, distended and there is constipation. The heart is tumultuous, the pulse small and weak, the urine scanty and loaded. Finally, there is some delirium and absolute loss of sleep.

Our first care should be to order balneation, methodically carried out and supervised, which will act upon the temperature, circulation, the secretions and nervous system. But at the same time antisepsis of the natural cavities is to be accomplished, as they offer excellent *nidi* for the creation of secondary infections. Watery vaporizations should be ordered to bring on sleep, calm the cough and fluidify the bronchial secretions. Digitalis is exhibited to sustain and regulate the heart, large quantities of watery drinks are to be taken to open up and flush out the kidneys and alcohol in some form as a general stimulant. The evacuation of the bowels is to be attended to and

from time to time a small oil purge is to be prescribed in order to obtain a true physiological antiseptis of the intestine.

And if, instead of an acute broncho-pneumonia, the pulmonary process assumes a slower type with morning remissions and evening exacerbation, quinine is indicated rather than digitalis, the former drug being, to my mind, the best remedy we have for the remittent rather than the continued type of the infection, and should the case be one with a distinctly localized lesion, with fine râles, one should not hesitate to apply, with all usual precautions, a blister, as it will render unquestionable service when it is applied *à propos* and not at *tout propos*.

Now let us take a case of cholera. Vomiting and diarrhœa are profuse. The heart is weak, the pulse small, the skin cold and clammy; the eyes are sunken, the face pinched. Collapse is evident. The physician should deal with each indication present. By an absolute diet the introduction of all toxins into the organism is suppressed, a cold, slightly alcoholic gaseous drink is ordered. For the vomiting Rivière's \* potion is prescribed. For the cold and depression very hot mustard baths, followed by energetic friction, find their indication. For the intoxication injections of physiological salt solution, to which a small dose of caffeine is added, are useful and only later on will the diarrhœa be attended to, because it eliminates the poisons from the body and consequently should not be interfered with too soon.

These illustrative cases show, I think, the way in which I regard the conduct of the physician should be and the manner in which infantile therapy should be handled.

With the *indications* there are always the *contra-indications*. The latter are often found in the patient himself. In children some pre-disposition, certain conditions of the functions of various organs or

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\* NOTE.—Rivière's potion is composed of two mixtures:

Mixture No. I.		Mixture No. II.	
R		R	
Sodii bicarb . .	3.5 grammes	Acid citric . . .	4 grammes
Aq. dist. . . .	100 c.c.	Aq. dist. . . .	100 c.c.
Syr. sacchar. . .	30 c.c.	Syr. acid citric.	30 c.c.

A spoonful of the first mixture is given and is immediately followed by a spoonful of mixture No II. Largely used in France and very efficacious. (*Translator.*)

some complication will contra-indicate a given drug or medication. Antimonium sulphuratum should not, for example, be given to a child with gastric irritation or diarrhoea. A typhoid case with nephritis cannot be benefited by cold baths, while in scoliosis, digitalis will quickly paralyze the heart which is compressed by the spinal deformity. And it should be recalled that the gastro-intestinal infection which exists in a latent state in 80 per cent. of children frequently will constitute a substratum of the highest importance, which must be reckoned with if mistakes are to be avoided.

But the majority of the contra-indications are to be found in the *remedy* itself. For years it has been common knowledge that certain drugs do not agree with children, and it is hardly necessary to recall opium and its derivatives in this respect, although I fancy that their dangers have been somewhat exaggerated by some observers. It is generally maintained that in children absorption takes place quickly and easily and elimination likewise, so that there is no reason why active remedies should not be exhibited on the condition that they are prescribed in small doses given at infrequent intervals. It is upon the express condition that the liver and kidneys are functionally normal, which is not the case in the major infectious processes with high elevation of the temperature, or in those subjects who have "weak bowels" and those who easily develop septic processes. I had a very curious case at one time of death unquestionably due to the immoderate use and too long continued of iodized absorbent cotton. It was a child with broncho-pneumonia of moderate intensity. The attending physician had the unfortunate idea of covering both the entire back and chest with this cotton, over which a layer of gutta-percha was placed. The urine became scant, albumin appeared and the little patient promptly died of uræmia.

Children's skin is, in fact, very susceptible to drugs. It does not tolerate carbolic acid, mercurial ointment, etc., which are prone to give rise to erythema and subsequently to erosions and ulcerations which serve as open doors to infection. Mercurial preparations exhibited internally usually act badly unless given for some special indication. Internally, strong antiseptics, naphthol and its compounds quickly cause constipation and diminish the urinary secretion. Even boracic acid should not be given over a too-long period in the form of enemata, because it is absorbed and is toxic, although



not in a high degree, it is true, but nevertheless it is certainly so. Caffeine is largely employed in pediatric practice, at least in France, but it should never be prescribed in large doses and not at all in acute diseases with a high temperature because of its exciting action on the nervous system, which, in childhood, is so sensitive to all stimuli. It should be kept for cases of collapse. Finally, odoriferous medicaments are not readily accepted by very small children, and I think that the iodine compounds should be excluded in infants, as their wounds can be dressed with perfect success with subnitrate of bismuth or the subcarbonate of iron.

Therapy is another thing than a reduced nosology. The doses should not merely be appropriate for the age and weight, but above all to the circumstances and conditions offered in a given case—in one reduced, in another normal, in still another greatly increased. When necessary, the practitioner must be audacious if the situation requires boldness on his part.

As to the form in which to give drugs and the manner of their administration I shall not take up the reader's time, as the subject is too well known and commonplace to require comment. Let me only say that not having to deal with beings endowed with reasoning power the physician should avoid disagreeable or repugnant remedies to young children. Tinctures, when carefully prepared, are very practical and easy to handle. Subcutaneous administration of drugs is becoming every day more popular in pediatric practice and more and more extensively resorted to.

He who would believe that infantile therapy only comprises the *diseases* of childhood and the description of medications will be grossly deceived. Quite on the contrary, it should be widened to its utmost extent and be regarded as the *ensemble* of measures to be employed for assuring the normal and regular development of the child, to prevent disease arising in him by modifying his pathologic temperament and even to make over the normal temperament. To administer drugs is not all; hygiene, prophylaxis and the rearing of children are of quite as much interest to the physician enjoying a children's practice.

Children are more apt to be ill on account of infraction of the laws of nature and of hygiene committed by the parents or because they were ignorant of the means to prevent this occurrence. How

are such infractions to be prevented? How can young or older children be made to observe these laws? It seems to me that by development of their understanding or, as it might be called, *peuriculture*, is the best means. It is hardly possible to believe that even to-day, after all the work that has been carried on by various philanthropic workers the foundation of societies for the relief of mothers and children, infantile mortality is relatively high, in spite of the diffusion of knowledge of elementary hygiene. A well-developed infant, born at term, well nursed and well nourished, should not die, but just here lies the difficulty. The children of the poorer classes rarely or never realize these conditions. When they are breast-fed, it is the mother or the wet-nurse who alone acts, and usually they are guided by the prejudices of ignorance. The infant is only shown to the physician when he is ill—often dying.

If the infant is bottle-fed the mother never dreams of asking professional advice as to the quantity or quality of the milk to be given according to the nursling's age and weight. No attention is paid to the regularity of the feedings or to those precautions so essential relative to changes taking place in the milk. The gastro-intestinal dyspepsia becomes silently established, which is made evident by foul, infected stools to which no attention is paid by the parents and then some fine day a cholera develops. Then it is too late.

The contagious diseases, eruptive fevers, do not usually attack the newly-born or older infants unless they are brought into direct contact with the focus of contagion, so that if they are kept some distance from this focus they are likely to remain intact. This prophylactic measure should be carried out in every instance, because from its simplicity it is easy to execute.

The animal races and stocks are constantly being ameliorated; the hygiene of stables, etc., is being carried out extravagantly and at great expense, but many families—farmers especially—hesitate to pay out money for the rearing of their offspring, which unquestionably is a part of the wealth of their country. The state, cities and towns should establish modern dairies where milk can be bought at as low a price as possible and where it can be thoroughly supervised. Many other conditions along these lines could be considered, but space does not permit.

Another function of the physician is to superintend the weaning of infants. How many disasters would be avoided if a competent man had the direction of this difficult transition in charge. Besides the victims of milk there are the victims of feeding. How many enteritides could have been avoided or promptly controlled if a proper selection in the diet had been indicated in time. But if it has been impossible to prevent the worst from happening, it is then the duty of the physician to attempt their repair; although it is impossible to make over the adult viscera anew, it can at least be hoped this result may be obtained in children who are still in the state of evolution. This is the *therapy of regeneration*.

Like all other practitioners who see much of children, I have very interesting notes of infants bottle-fed and brought to hospital in a deplorable state of athrepsia, atrophy or dystrophy. By properly supervising the feedings with a proper dosage of sterilized or pasteurized milk and a little hydrochloric acid to help digestion, a real resurrection is brought about in the condition of the infant. Other children who have been weaned for some time have been reduced to the state of a skeleton from gastro-enteritis. After controlling the diarrhoea, which can often be accomplished by stopping cow's milk, which is really impossible to digest, the dyspepsia—the fundamental cause of all the trouble—is treated and by prudent and rational feeding the patient can be brought back to a normal state; hot salt baths and a cure in the open will achieve a cure which seemed improbable at the onset. The body weight increases, the skin takes on a life-like look, the liver decreases in size, the pulse and heart become strong and the child's spirits return. There is no doubt but that repair is evident and complete.

In somewhat older children mountain climate and sea cures may be utilized with advantage. I am convinced that children obtain the greatest benefit from hydro-mineral medication in overcoming their hereditary or personal defects. This medication is easier to regulate, less dangerous than sea cures, which are too violent for young subjects, who offer a sharp nervous reaction. Here, too, there is a choice to be made which the physician alone can make after examination of the little patient.

To sum up my thoughts, I would say in conclusion that the various ways of regarding infantile therapy seem to me all useful and that

the best method—if method there be—consists in endeavoring to obtain the conditions of a *rational plan of therapeutics* as complete as possible in the light of our present knowledge, and it might very properly be called *scientific or clinical therapeutics*. Its object is to enlighten us—after a minute examination—on the *indications* derived from the examination and pathogenesis of a given case, as well as the contra-indications. Infantile therapy will only merit and justify this name upon the condition that it is both prophylactic and regenerating; in other words, the child must be built or rebuilt as the case may be.

## ILLUSTRATIVE CASES OF STAPHYLOCOCCIA IN PÆDIATRIC PRACTICE

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THIS paper is not written with an intention to add anything new or scientific to the subject of staphylococcia, but is merely intended for the general practitioner who is likely to encounter cases in practice among children. One is often struck by the number and at the same time the diversity of affections caused by the same vulgar microbe in childhood. From the special viewpoint with which this paper is concerned, three orders of pathologic evil can be laid at the door of the staphylococcus: (1) superficial infections, (2) deep-seated infections and (3) the transformation of these infections into each other.

I have selected from my notes the more common cases as they occur in our daily work, and I repeat that I have no intention of offering a deep or complete study of staphylococcia. The staphylococcus presents three types; the aureus, albus and citreus. They belong to a single species and are distinct only from their chromagen substance. In fact, the staphylococcus aureus can be artificially prevented from producing its chromagen substance by depriving it of oxygen or by adding antipyrine to its culture media or exposing it to the solar rays. I would observe, however, that these changes are only temporary, because as soon as the organism is replaced in normal conditions it takes back its color. However this may be, the staphylococcus is one of the most widely-distributed pathogenic bacteria; it is met with in the air, in water or in ice, where it can live for a long time. It is the usual occupant of the natural cavities of the body and even of the viscera. Vigual pretends that it is more common in the mouth than the leptothrix, while Létienne has often found it in the bile.

Its presence is still more constant on the surface and in the depths of the skin, but its frequency varies with the manner of life of the subject, as well as the region of the body; that is to say, it is found much more easily in an uncleanly person and on those parts of the body the most exposed.

The staphylococcus resists a temperature of 50° C. for not more than forty-eight hours; solar light and electric rays quickly diminish its virulence and change its chromogen power, but contrary to other bacteria, a pressure of ninety atmospheres has no effect upon either its vitality or its virulence. Iodoform has no action upon this organism, while carbolic acid, Hg. bichloride, Hg. biniodide and copper sulphate are the antiseptics most apt to destroy it.

The staphylococcus offers the peculiarity of simultaneously manufacturing in the culture broth antagonistic substances, some vaccinating, the others predisposing. It secretes pyogenic toxins and vaso-dilating toxins. All these products, both soluble and insoluble, exist at the same time in the cultures and are reciprocally influenced. But if they are separated by filtration, a principle remains in the filtrated liquid which when inoculated immediately brings about cardiac weakness, a drop in the temperature and some convulsive attacks.

Unquestionably, it is due to the diversity of the properties of its products of secretion that the staphylococcus produces such multiple lesions in the human body, appearing in such varied clinical forms that at first sight they do not seem to belong to the same nosological family. However, when all these affections are studied, one will soon perceive that they are bound to each other by many bands of union, the principal pathogenic agent being the staphylococcus.

The infection may be produced by an autochthonous staphylococcus, living as a saprophyte on the subject who becomes its victim, or by a staphylococcus brought by contagion. A simple, inoffensive saprophyte staphylococcus may at certain times acquire pathogenic properties. As in the case of all infections, the seed and the soil in which it develops must be considered, the part played by the soil being all-important. We know that the virulence of certain bacteria is increased by repeated passage from one subject to another. For that matter, this is a procedure employed in bacteriology for increasing the virulence of bacteria in general.

Now, this procedure accidentally finds its application in the human organism, and I will give an example. A nursing, by producing fissures of the nipple by sucking infects the mother by inoculating with the saliva staphylococci, which until then were inoffensive for the infant, but which in the mother give rise to a galactophoritis and abscess of the breast; that is to say, a production of pus. This pus,

afterwards absorbed by the infant, causes a diphtheroid stomatitis or still more serious lesions.

It is, therefore, clear that the staphylococcus, perfectly harmless in the infant's mouth, increases its virulence by passing into the maternal organism. Another proof of this fact are the epidemics in hospitals which sometimes appear, in which a mere crust of impetigo due to the staphylococcus will give rise to vulvo-vaginitis, otorrhoea or intestinal and broncho-pneumonic infections whose gravity increases with the number of cases.

According to some observers, the importance of the morbid soil is far greater than that of the germ itself, and of all the general causes the age of the subject stands first. While in the adult affections produced by the staphylococcus are in most cases mild, they can become very serious in children. It can almost be said that the resistance of the organism to the staphylococcus progressively increases with age, and although puny children die from this affection practically without exception, healthy and robust children resist, and resist all the better the older they are.

Rodet long since demonstrated perfectly that if the same lesions produced by the staphylococcus in children were to be reproduced in animals, these should be selected among the more sensitive—the rabbit, for example—and also to choose a young subject in the full period of growth. In fact, growth predisposes the organism to infection from pyogenic organisms and favors their localization in the ends of the long bones particularly.

Acute and chronic diseases are also conditions to be taken into account. The staphylococcal infections are not uncommon in children after serious diseases, such as typhoid, influenza or pneumonia. They are frequent in those afflicted with chronic processes, such as gastro-enteritis and dilatation of the stomach. The same applies to all debilitated children or those issued from elderly, alcoholic or syphilitic parents.

But there is an affection whose predisposing influence predominates all others—I refer to tuberculosis. The lesions caused by it are often overlooked during life, but they are frequently found at autopsy in children dying from a staphylococcal infection whose pulmonary symptoms and cachexia had been attributed to the staphylococcus. It is not uncommon to meet with an association of this bacterium with

the bacillus of tuberculosis, each one acting on its own account or combining their action in one. Here is a case in point:

G. S., *st.* three months, a pale, puny, thin infant, only weighing seven pounds in spite of its age. The patient coughs; auscultation reveals subcrepitant râles. The integuments are covered by cicatrices, remnants of a former cutaneous infection. Over the buttocks and anal region there is a rather marked erythema over which some eroded papules are scattered. The folds of the buttocks are rather thin. The stools are mixed, there being four daily; vomiting. Temperature, 101.5° F. The child was put on the bottle and the stomach flushed out. On the following five days the temperature dropped and she gained weight.

On October 15th, vomiting and diarrhoea returned and patient lost weight. Irrigation of stomach and bowel; lime-water internally and subcutaneous injections of artificial serum.

The sacral region and external iliac fossæ were dark red and erythematous. The eruption does not reach the anus, but extends down the thighs; it is limited by a distinct margin but not raised. In the popliteal space and over the calves are two small erythematous areas, each with a purulent vesicle in the centre. Fearing the advent of an erysipelas, a 1:2000 sublimate-ether spray was used on the margins of the erythematous areas.

October 20th: The lymphangitis has not extended; bacteriological examination of the contents of a vesicle gave pure cultures of the *staphylococcus aureus*.

October 28th: The general condition is worse. The diarrhoea has increased and the stools are green; emaciation has progressed. The temperature is up, oscillating between 102° to 104° F. A few scattered râles on auscultation.

October 29th: Numerous subcutaneous abscesses have developed with pustules on the lower limbs, shoulders and posterior aspect of the scalp. The abscesses are incised and dressed antiseptically, but they have little tendency to heal; they give rise to undermining of the skin with sinus formation.

October 31st: More abscesses have appeared on the back, buttocks, thighs and popliteal spaces. From this time on the child gave up the struggle, became very pale and emaciated; the temperature



progressively fell. On November 8th it was 98.6° F. in the morning, 97.5° F. in the evening. The child died during the night.

Autopsy on the following morning. The lungs were absolutely riddled with miliary tubercles over the surface and in the pulmonary parenchyma. The lower lobes are in a state of high congestion. At the lower part of the right lung was a whitish mass having the aspect and consistency of a raw chestnut. The apex of the right lung was adherent to the costal pleura. The lymph-nodes of the mediastinum, neck and groin are caseous. Under the parietal pleura, in the intercostal spaces on the right, tuberculous lymphatic chains could be seen. Although bacterial examination of the pus from the abscesses was done upon several occasions during life, only the staphylococcus aureus could be grown.

No doubt can be entertained about the coexistence of the staphylococcus and the tubercle bacillus in this case. Claisse has reported a case of cardiac malformation which seemed to have favored the development of a fatal staphylococcia in a girl of nine years. Nor is this all. The external conditions must be equally taken into account—the environment in which the children live. The privation of air and sunlight have long been known to exercise a very great influence on the resistance to infection of the human organism. The lack of cleanliness and other care of the body plays a double part, as it causes a predisposition to infection and at the same time brings the necessary germ for this to take place. Premature solid feeding or undigestible foods—in a word all the deplorable conditions which engender physiological misery in children in large cities or towns—are all so many indirect causes of staphylococcia.

Regardless of the amount of study lavished on the subject, the mechanism of staphylococcal infection—its penetration into the organism—has not been made perfectly clear. Many theories have been put forward and the contradictory findings are to be explained by the fact that the mechanism is not a single one but must very often vary. It cannot be denied that dried septic bacteria can be transported by inhalation to the respiratory tract or absorbed by the digestive mucosa.

Thus can undoubtedly be explained some of the cases of bronchopneumonia which occur during a staphylococcal infection and frequently end in death. It is also in this way, perhaps, that may be

explained the development of certain intestinal disturbances which occur as a secondary complication. Other observers admit that the staphylococcus passes directly into the digestive track and in the blood. It may, in reality, happen that the gastric juice is mildly acid and does not kill bacteria, which will then develop easily in the alkaline midst of the intestine.

This theory which rather readily explains the production of multiple abscesses has been sharply contradicted by Hulot, who does not believe in this manner of penetration or at least regards it as very exceptional. According to Hulot, the multiple abscesses observed in nurslings are simply the result of cutaneous inoculation in children presenting a cutaneous lesion of the face or buccal mucosa, all the more so, he says, because the subject dies soon after the penetration of the staphylococcus into the blood.

It is true that, when histologic sections of the skin are examined, it will be seen that the capillaries situated below the purulent focus contain masses of staphylococci, either free or contained in fibrinous coagulations; but it is not impossible that the staphylococci encountered at these spots may have been brought there by the circulation.

Hulot's very exclusive opinion is not sufficient for always explaining the production of deep-seated abscesses and general complications. However, the skin is an organ which reacts like any other organ when in presence of a morbid agent. It can react at the point of attack or the reaction may take place elsewhere. But if this manner of infection be admitted, how is the manner of penetration of the staphylococcus into the dermis to be explained?

When the skin is healthy, the epidermis offers an impassable barrier to staphylococcal penetration and then one might be led to suppose that the organism penetrates by way of the excretory ducts of the glands—sebaceous or sudoriparous—or by the hair follicles. But the staphylococcus has been rarely encountered in these localities.

If the horny layer has undergone a change nothing is then easier. The staphylococcus, like all other bacteria, travels directly in the interval existing between the cells of Malpighi's mucous body and thus arrives, free or included in leucocytes at the surface of dermis.

This opinion, whose logic has been demonstrated by Bockart, by placing cultures of staphylococci on his arm after having rubbed the horny layer was maintained long before him by Escherich. For that

matter, the cause of changes in the epidermis are not by any means wanting in infants and children any more than they are in adults. Among the most important of these causes are lice, far more common than generally suspected. They produce severe pruritis, and by scratching, which is the consequence, the nails produce solutions of continuity of the integument through which the staphylococcus enters. Then came eruptions of all kinds which, opening on the exterior, produce changes in the skin. Finally, some observers have insisted upon the small ulcerations which children wearing diapers have when this article of infantile apparel is not properly attended to and is left moist with urine and feces in contact with the skin.

However this may be, whether infection takes place in one way or another, it is none the less certain that the staphylococcus enters the lymphatics and is then rapidly carried by the lymph to the lymph-nodes in which it almost always sets up an inflammatory process and occasionally a suppurating adenitis. Once in the lymph-nodes it is arrested for a time during which it may become attenuated or destroyed by the phagocytes, or else it escapes destruction and continues onward only to exercise its baneful action.

I shall now consider some of the staphylococcal affections and will begin with impetigo. This is an affection characterized by superficial pustules deprived of any peripheral inflammation, followed by the production of yellowish crusts which fall off without leaving a cicatrix, unless the pustules have been excoriated by scratching. The majority of observers admit that impetigo is produced by the staphylococcus, as this organism has been constantly found in the contents of the pustules and I am of this opinion.

Impetigo often coexists with other superficial suppurative processes in children, such as subepidermic whitlow, vesiculo-ulcerative lesions, conjunctivitis and stomatitis; all these lesions are produced by the same pathogenic agent. Impetigo is met with especially in children, particularly in lymphatic subjects who offer an excellent soil for the development of pyogenic bacteria.

Ordinarily the eruption occurs all at once. In these circumstances a slightly pruriginous redness appears, followed by a little, round, yellowish pustule distended with purulent fluid; other pustules afterwards develop, remaining isolated or becoming grouped in more or less extensive clusters. They vary in size from a hemp-seed to a lentil.

By scratching they burst, giving issue to a yellowish fluid, which dries and forms soft, friable crusts. Sometimes the crusts become blackish from excoriations which have produced a slight hemorrhage.

The crusts drop off spontaneously or by treatment, but the skin remains red and secretes a liquid which gives rise to fresh crusts which become thinner and thinner until at last they disappear. Impetigo is very common. It is eminently contagious and spreads by simple contamination or, as in other staphylococcal infections, by transformed contamination.

Male, *æt.* two years. Impetigo of the face and head, dating back for several days; vesicles on the left ring-finger. The contents of the pustules gave cultures of *staphylococcus aureus*. Became infected by kissing his mother, who had one pustule on the face.

Male, *æt.* twenty-two months. Father died from tuberculosis. The baby was weaned on October 22nd, the day he entered hospital, with a diagnosis of impetigo. There were crusts on the scalp, face, cheeks and arms. The lesions were treated and progressively disappeared.

On November 10th, a small tumor was observed on the palpebral conjunctiva the size of a hazel-nut and having the aspect of a gumma. The tumor was a red-violet color and fluctuating; pressure on it did not give rise to pain. Bacteriological examination gave a pure culture of the *staphylococcus*.

November 19th, this abscess had increased in size and gave issue to pus mixed with blood. Enlarged lymph-nodes were palpated in the neck.

November 21st: Severe diarrhœa, in larger quantity in the morning than evening. The stools are whitish. The abdomen is large and distended; there is an ulceration over the last dorsal vertebræ which does not discharge much if any.

November 22nd: Diarrhœa is a little less and the stools have become dark. Temperature, 96° F. in morning, 97° F. in evening.

November 24th: Temperature is higher and at 4 P.M. it was 99.8° F., but the abdomen is not so large and the diarrhœa has decreased.

November 28th: Diarrhœa has completely disappeared and the child was taken home.

J. E., *æt.* three and a half years. Bottle-fed child; partook of solid food at thirteen months. When eighteen months old he had a

boil (?) on the scapula following the application of a plaster. This so-called boil had persisted for one year. One of his playmates, who was the bearer of several impetigo crusts told the patient that he (the playmate) would give him his disease and thereupon voluntarily inoculated the patient on the ear, after having loaded his nail with a bit of crust.

The staphylococcus often produces pseudo-membranous conjunctivitis, which differs from catarrhal conjunctivitis by the nature of the secretion and especially by the frequency of corneal complications. These complications make this ocular process extremely dangerous, although they may sometimes be cured by treatment or pass into a chronic state.

The evolution of purulent conjunctivitis is usually made up of three phases, the first is characterized by redness, heat, tumefaction and pain. The infection begins in the palpebral conjunctiva, the patient experiencing pruritus and a sensation of great dryness with an evident elevation of the temperature of the lids.

During the second phase the characteristic pus secretion appears, and at the same time the conjunctival tumefaction increases in considerable degree. The upper lid forms a rounded, hemispheric, red and shiny projection which partially overlaps the under lid. The secretion occasionally produces a high degree of irritation of the cheek, while the pus has a tendency to collect especially in the greater angle of the eye. This secretion, which is intensely virulent, will reproduce the same process when inoculated on a healthy eye, so that it is not uncommon to meet with cases in which the eyes are successively involved. There is a mild degree of photophobia. The process may become spontaneously arrested at this phase, in which circumstances it must be inferred that the soil has become unfit for the development of the staphylococcus. But such is not always the case, and the process goes on to its third phase.

At this time the pain, which had decreased when the suppuration appeared, again becomes acute, and shooting, extending around the orbit in the fronto-temporal region. The tumefaction of the lids persists, as well as abundant production of pus; at the same time, some ulcers may be noted, or infiltration and total necrosis of the cornea may occur, accidents which often complicate pseudo-membranous conjunctivitis.

This affection, very common in children, is particularly prone to occur in those having impetigo or who live among other children presenting other forms of staphylococcic infection. It is especially by infected hands that the pathogenic agent is brought in contact with the conjunctiva or transmitted from one individual to another.

Female, æt. two years, was seen on August 12th, at which time she was treated for rickets. On November 12th the child developed an impetigo on the left ear following a scratch. A lotion of boracic acid was ordered and the treatment carefully followed, but this did not prevent the infection from reaching the eye on the same side and two days later a conjunctivitis became declared.

November 15th: The lids are very cedematous and much pus was discharging. The ocular conjunctiva is very hyperæmic; the palpebral conjunctiva presents false membranes and bleeds when these are removed. Cultures on blood-serum made to-day at 11 A.M., because diphtheria of the eye was suspected. The child was isolated at once. In the evening the temperature was 99° F., the pulse 144.

November 16th: Morning temperature 98.8° F., pulse 112. The culture on the serum did not develop. A false membrane, less adherent, lined the cul-de-sac. Another culture was taken. The patient's condition was satisfactory; he was playful and began to open the eye. Urine normal. Although the day had been good, it was decided to give a prophylactic injection of 10 c.c. antidiphtheritic serum. Temperature, 99.3° F.; pulse, 152.

November 17th: Patient was very restless all night; a sedative potion had had no effect. Cultures did not give any Loeffler bacilli. Morning temperature, 99.3° F.; pulse, 112; evening, 98.8° F.; pulse, 144. The day was good. Pus still discharged from eye; no false membrane; patient can see with diseased eye.

November 18th: The cultures taken on November 16th showed a rich growth of staphylococci. A few days later recovery was complete, although the lids were still somewhat red.

Vulvo-vaginitis due to the staphylococcus is not uncommon and is met with both in hospital and private practice. The process is a very intensely inflammatory one. The labia majora are swollen and tense; the mucosa of the vulva and vagina is hot, red and covered with pus. Ulcerated follicles and papillary elevations will be seen and bleed very easily.

The vagina and vulva are the seat of a profuse discharge, occasionally with a foetid odor, greenish in color, spotting the linen heavily and producing excoriations of the region around the vulva. The little patients complain of pruritus, heat and burning in the vagina and a feeling of weight and pain in the perineum. They have a frequent desire to urinate and suffer from severe smarting during micturition. The lymph-nodes of the groin are enlarged and become painful; they may suppurate. These cases of staphylococcal vulvo-vaginitis are of extreme importance from the medico-legal standpoint, as they may very well be mistaken for a gonorrhoeal process.

Female, set. four and a half years. Two months before the present illness she had been submitted to digital manipulations by one of her little friends who had patches of impetigo on the face. The child had also slept with her grandmother, who had a leucorrhoea, on several occasions. The onset of the affection occurred at about Christmas.

The child complained of some pruritus about the vulva and experienced some difficulty in micturition. In looking the child over, the mother noticed a greenish discharge and concrete pus on the labia.

The patient was seen for the first time on January 7th, and the existence of a vulvo-vaginitis was noted. Bacteriological examination gave a pure culture of the staphylococcus aureus. Irrigations with a 1:4000 sublimate solution were ordered and salol bougies. This treatment resulted in recovery in a few days.

Female, set. four and a half years, was seen on December 1st. She presented vesico-pustules on the buttocks and labia majora and a vulvo-vaginitis. Cultures made from the discharge gave a pure staphylococcus growth. Sublimate irrigations for vulva and vagina and a boracic acid ointment for the pustules. The patient was completely cured on December 20th.

Although the staphylococcus is more prone to be associated with pultaceous angina, it can also give rise to pseudo-membranous angina in certain circumstances, especially during infectious diseases. These are not rare and in some cases attain a certain degree of gravity, as I shall show by a case report.

In these pseudo-membranous anginas both the staphylococcus albus and aureus are found. The propagation takes place by the usual means; in the first place by autoinoculation—patches of impetigo on the body followed by angina—or by simple contagion or transformed

contagion, the child living with other children having the same lesions or lesions due to the same etiological agent: impetigo, conjunctivitis, superficial suppurative processes, etc.

This affection manifests itself by an inflammation of the mucosa of the fauces, at first superficial, but which is not long in becoming deep-seated and accompanied by exudation in which the staphylococcus is constantly found. At times localized, it occasionally extends over the entire surface of the fauces and pharynx; while in some cases the general symptoms are very mild, in others they may acquire such intensity that they lead the physician to suspect the advent of some very serious process and may even give rise to laryngeal accidents simulating diphtheritic croup. The following case is a good illustration. It will be observed that the subject also had a purpuric erythema, probably due to the same causal factor and enters into the category of infectious erythemata to which so much attention has been given.

F. J., *æt.* two and a half years. In June had been treated for a staphylococcal laryngitis. Seen again on January 15th of following year, at which time the child had been coughing for a fortnight. Very weak and hoarse. Respiration almost normal, throat red; a few minute false-membrane on pillars; very marked submaxillary adenitis. Below the ear on the left cheek was a purpuric erythema in pin-points. No albumin in urine. A culture taken at the time had not given any growth the following day. Prophylactic injection (10 c.c.) antidiphtheritic serum. On the second day a pure culture of staphylococcus had developed.

January 17th: Same condition. Patient slept poorly; numerous vomiting attacks. In the evening of the 16th, four hours after the serum injection an eruption of urticaria appeared. No albumin in urine.

From January 18th to 21st the condition improved; sleep quiet; no albumin. On January 21st there occurred a second eruption of urticaria starting at the spot where the injection had been given.

January 23rd: A fresh eruption of urticaria invading the face, abdomen, back and legs and left sufficient swelling to simulate an cedema. No albumin.

January 24th: Tongue coated. Castor oil. From this time



on improvement continued and the patient was discharged well on January 29th.

The staphylococcus does not always limit its action as in the preceding processes. It often produces vesico-pustules scattered all over the body, a true generalized superficial infection which may very properly be termed *superficial staphylococcia*. This infection, like all others due to this bacterium, is contagious and develops rapidly in children, especially when lesions of the epidermis are present. The following case is illustrative:

Male, *æt.* ten years. Had had typhoid fever the previous summer. The boy, at boarding-school, played frequently with one of his play-mates who had some vesico-pustules on the face. About the commencement of October he experienced pruritis over the entire body and large "pimples" appeared on the chest and back; they then extended and reached the neck, ears and thighs; the legs were the last to become infected.

January 10th: When seen to-day for the first time, cicatrices were noted between the shoulder-blades. Many confluent vesico-pustules are seen on the left hand and arm, especially over the wrist and dorsal aspect of the hand. The majority of the pustules have burst and emptied their contents. The right hand and arm are not so involved. Both thighs present the lesions over their posterior aspect.

Bacteriological examination gave a pure culture of staphylococcus albus. A boracic-acid lotion was ordered and moist dressings applied. Sulphur baths once daily. When the patient remains for a few hours without the moist dressings on the head, the pustules seem to revive anew and those on the ears suppurate again. The boy was fearfully troubled with pruritis and scratched off the crusts with the nails.

January 29th: Some traces of prurigo are seen which were suspected to be due to scabies, but two days later he had recovered perfectly so that he could be discharged.

I have now considered some of the more common infections due to the staphylococcus as met with in daily practice, but there remain to be mentioned the complications of these infections, as they are no less interesting and important. We know that all these infective processes can be propagated by similar contamination or transformed contamination. In the first place, every child with a staphylococcal infection of any type may present lymph-node involvement in the

region of the lesion or at a more distant region. When tumefaction of the lymph-nodes of the region, the site of the lesion, occurs this is perfectly natural.

The cervical and submaxillary lymph-nodes are most commonly the site of impetiginous infection, because the face is the point of election of impetigo. In vulvo-vaginitis likewise, the lymph-nodes of the groin—being nearest the lesion—are the first to be involved; when the tumefaction of the lymph-nodes of the involved region takes place there is nothing surprising if the lymphatic glands of a nearby region become infected in their turn. Clinically, this often occurs, and since the gland tumefaction may extend gradually there is no reason why quite distant areas should not become involved. Here is a case in point.

Male, æt. two and a half years. Breast-fed. A pertussis-like cough has been present for almost a year past. The child presents vesico-pustules scattered over the chin, which gave pure cultures of *staphylococcus albus*. A severe impetigo of the face developed, this being very shortly followed by a multiple polyadenitis.

The adenitis is remarkable in that it is exceedingly persistent, and very frequently children who have been cured of their lesions for months will still offer enlarged lymph-nodes. These are unfortunate occurrences, not merely because of their tenacity, but on account of the interminable suppuration to which they not infrequently give rise. It is easily comprehensible that the *staphylococcus* when once securely lodged in the organism, may give rise to numerous complications, some being mild throughout, while others may take on a certain gravity, such as multiple abscesses often met with in nursing infants, subcutaneous phlegmon and phlegmonous tonsillitis.

And yet this is not the end of the list of complications. The *staphylococcus* may enter the joints or bone marrow, where it provokes suppurating arthritis and that formidable affection of childhood—acute osteomyelitis. Carried by the blood-current the organism reaches the heart where it provokes a rapid form of infectious endocarditis.

The respiratory complications due to the *staphylococcus* are not less serious. Not only is this bacterium to be found in the sputum of bronchitis and bronchial dilatation combined with other organisms, but the products expectorated in gangrenous bronchitis contain it as

well. Both the albus and aureus produce tonsillitis and fatal croup and bronchopneumonia.

Male, set. thirty-one months. First seen June 2nd. Both parents are in good health. Child was breast-fed. Pertussis at the age of eight months; at twenty months had a sore throat, since which time the infant has been frequently ill. For the past few days has been restless with headache and abdominal pain. The mother noticed a swelling on the left side of the face at the angle of the jaw. Child swallows with difficulty.

Examination shows a tumefaction at the angle of the left jaw occupying the entire region and extending upwards to the parotid. The mass is hard, movable and painful. On the surface enlarged and inflamed lymph-nodes can be felt. No cough; lungs negative.

The tonsils are tumefied; some white spots the size of pin-heads are seen on their surface, especially on the right tonsil. Voice clear; no difficulty in breathing. Temperature oscillates between 101° and 102° F. Constipation; tongue very coated.

June 4th: Respiration calm; no cough. Two stools in morning. Throat in the same condition. Voice slightly hoarse. Normal temperature. No albumin in urine. Culture taken from tonsils gave a pure staphylococcus growth.

June 5th: Night good, sleep calm. No cough or constipation. Voice clearer. Tonsillitis cured; adenitis less. Discharged.

The digestive apparatus may be infected by the staphylococcus, likewise the salivary glands and pseudo-membranous angina, as in the case just reported. The buccal mucosa may become involved by an extension of an impetigo of the face or by contagion if the infant has ingested pus contained in the maternal milk. A diphtheroid stomatitis results.

Fissures and easily bleeding ulcerations occur on the lips which are tumefied, painful and covered with brownish crusts. Then these ulcerations attain the internal surface of the lips and form whitish patches having a diphtheroid appearance. They usually retrogress in about ten days' time and the child recovers if they do not cause some other focus of infection elsewhere in the organism.

The buccal infection by extension produces tonsillitis or parotiditis which, in their turn, may represent the initial focus of a general

infection. Retro-pharyngeal abscesses may be one of the primary or secondary foci of the infection.

A gastro-enteritis is frequently a staphylococcus infection in children, offering all degrees of gravity. Acute gastric ulcer has been observed in a case of generalized staphylococcus infection by Widal.

Staphylococcal infection may give rise to miliary abscesses in the intestine seated in the thickness of the walls of the gut. Serous peritonitis and ovaritis are not uncommon complications of staphylococcal vulvo-vaginitis. The liver and spleen may be the seat of deep abscess in the organ. From the pharynx this bacterium may reach the middle ear, producing suppurating otitis, which in turn may result in deafness. The brain may become infected and abscess of the cerebral sinuses and fatal meningitis are the consequences. The genito-urinary apparatus, on account of the considerable part it plays in the elimination of bacteria, is also generally involved. Small, miliary abscesses of the kidney are often observed; they are rounded in shape and on section of the organ they form a triangle with the base at the periphery of the organ, projecting on its surface. But nephritis is the most common process of renal involvement from the staphylococcus in both children and adults and in some instances it may overshadow all other symptoms of the infection; but generally it is transitory and only gives rise to a slight albuminuria, such as is met with in almost all acute febrile processes.

Male, æt. four years. Seen June 23rd. Breast-fed up to the age of two years and six months. Last year had an attack of impetigo which lasted several months. A fresh infection showed itself a fortnight ago. These two attacks of impetigo occurred as a consequence of injury to the hand, but the eruption commenced by the face.

On July 4th the child developed anasarca and the urine contained albumin in large amount, but completely disappeared on July 5th and 6th. The anasarca disappeared on July 15th. The child was seen on August 8th, in good health. Urine negative.

To sum up: All staphylococcal infections may result in various complications, the principal ones being: In impetigo there is almost always a regional adenitis which may or may not suppurate. Pseudo-membranous conjunctivitis may give rise to blepharitis, ulcers and

total necrosis of the cornea, the most serious consequence being the loss of sight.

Staphylococcal vulvo-vaginitis may extend to the uterus, tubes and peritoneum. Pseudo-membranous angina can extend from the pharynx to the middle ear, causing suppurating otitis. All these local processes may be the starting-point of superficial general infection or a fatal septicæmia. In children staphylococcal infection assumes four principal forms: multiple abscesses, hemphigoid and pustular eruptions, lymphangitis and disseminated cutaneous gangrene.

Multiple abscesses arise either in the skin or subcutaneous connective tissue. The superficial abscesses have two favorite sites, the scalp and the face, and secondly, the buttocks and perianal region. In fact, in children these regions are the most exposed to inoculation. They begin by a red-violet color of the skin, which is raised with a diffuse base, slightly hot and tense, but not very painful. The lesion increases and points to the surface, the apex soon assuming the form of a small pustule which opens, giving issue to much thick pus coming from the depths of the skin. Then the abscess collapses and rapidly heals. These abscesses vary in size from a small pea to a hazel-nut and in number they are considerable. Their repetition is often incessant and lasts for weeks or months. They appear in successive crops, but in these circumstances the general phenomena are serious and death may be the outcome, as in the following instance:

A pale, puny infant, weighing five pounds, born on December 10th, first seen on March 9th of the following year. A subcutaneous abscess on the back, followed three days later by one in the axilla.

March 30th. Eruption of pamphigus on trunk and limbs, also palms of both hands.

April 3rd: The first crop of eruption has desiccated. A fresh eruption appeared over the entire body. From this time on a series of abscesses appeared on the scalp, trunk and limbs, beginning with a hard papule, slightly elevated and which became acuminate, violet in color, surmounted by a yellowish pustule which enlarged and then suppurated.

From time to time the temperature reached 102.8° F., this coinciding with the formation of a crop of abscesses. Cachexia developed and the baby died on July 12.

Cultures of the pus made on several occasions always gave a pure growth of *staphylococcus aureus*.

Deep abscesses may develop at any part of the body but are more common in regions where the connective tissue is lax—around the anus, popliteal space, perivesical or perirenal areas. They are sometimes placed in series along the course of an inflamed lymphatic trunk, in which case they are true lymphangitic abscesses.

They begin as small, hard nodules, practically painless, projecting little above the surrounding cutaneous surface and are pinkish in color. They are better detected by touch than sight. They soon became violet and the region is the seat of a diffuse tumefaction. When left alone they enlarge and discharge, giving issue to a thick, yellow-green pus streaked with blood, often mixed with fatty detritus, but without traces of necrosed tissue. They are long in healing, cause extensive undermining of the integuments and coalesce with each other. They seem to represent a more advanced degree of infection than the superficial abscess and are also more frequently accompanied by high temperature, general disturbances and gastrointestinal phenomena.

Disseminated gangrene of the skin has been attributed to the *staphylococcus* by Charmay and Cailland amongst other observers. It usually follows impetigo, ecthyma and cutaneous staphylococcal abscesses, and when the liquid of the gangrenous bullæ is cultured a pure growth of the *staphylococcus* is always obtained. The following case is an example:

Female, æt. fifteen months. Measles on July 20th. A small cutaneous abscess developed on the head. Two days after the appearance of the eruption the temperature reached 104° F. and the bronchial râles increased. A whitlow on a finger suppurated and some small abscesses appeared on the scalp in the neighborhood of the initial focus.

On the following days the abscesses multiplied, reaching the neck and trunk, at the same time assuming a special aspect. A pustule having a pemphigoid look would just appear, rupture and give issue to a purulent liquid; the dermis being exposed continued to suppurate and became necrotic. The ulcerations, of gangrenous aspect, were surrounded by infiltrated, œdematous tissue having a red-violet color. The broncho-pneumonia declared itself and the patient's general state became serious.

July 28th: Temperature, 105.8° F. Abscesses increasing in numbers. The entire posterior aspect of the scalp is riddled by rounded, confluent wounds extending into the necrosed dermis and giving off a cloudy liquid. On the neck, shoulders, back, buttocks, and abdomen there were some pemphigoid pustules undergoing the same process of necrosis, resulting in punched-out ulcerations with a dirty discharge. The child died on July 31st.

Autopsy showed an intense pulmonary congestion with bronchopneumonia and a pleural abscess. No tuberculosis.

The pleural pus, the non-suppurating interlobar exudate and blood collected from the heart eighteen hours post-mortem, and the fluid from the pemphigoid bullæ, taken during life, gave pure and very virulent cultures of the staphylococcus aureus.

# Surgery

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## FECAL INCONTINENCE

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THE loss of fecal control may be complete or only partial. In transverse myelitis it may be mistaken by the patient for diarrhoea. A temporary partial incontinence sometimes is associated with the severe prostration of acute or chronic diseases such as typhoid fever, cholera, pneumonia, and tuberculosis. Incontinence in any of these conditions may be followed by impaction of the rectum because of the loss of tactile sensation of the rectum and the lessened secretion of mucus to lubricate the fecal mass.

The most frequent traumatic causes of incontinence of fæces are rupture of the recto-vaginal septum by childbirth, or traumatism to the parts adjacent to the operative field during rectal operations, particularly those for the relief of fistulæ and hemorrhoids and the improper divulsion of the sphincters for the relief of constipation and various reflex disturbances.

Another reason for partial incontinence is the adhesion of the mucous membrane to the fibres of the sphincter-ani, thus making it impossible for said muscle to contract or relax uniformly. This is the condition generally found responsible for ribbon-shaped stools or the uncomfortable sensation of an incomplete defecation.

Loss of sphincter control may be due to imperfect drainage, permitting the skin or mucosa to grow into cut; useless probing and cauterizing the wound. Division of the internal with the external sphincter does not materially increase the danger of incontinence, but severing the nerves controlling the sphincter does. Oblique, irregular or jagged incision of the sphincter, even though single, may result in such a vicious union of the muscle ends that incontinence



results. Transverse cutting of the sphincter is not likely to produce incontinence. The integrity of both sphincters is absolutely necessary for the voluntary control of the anus. If the muscle is cut squarely across and the ends united again so as not to materially increase the muscle length there will be good control. But if a wide band of scar intervenes between the severed sphincter ends the muscle will not sufficiently contract to thoroughly close the anus. Oblique incision of the sphincter allows the divided ends to slide upon one another until the internal fibres of one end unites with only the external fibres of the other end and thus very much lengthens the muscle and is frequently followed by incontinence. When the sinus of a fistula passes through the body of the external sphincter muscle an incision severs only the lower fibres. These may be left to close by granulations. But if the entire muscle is divided and the wound edges retract there is always some incontinence. If the sphincter is cut more than once incontinence is almost sure to occur.

Sometimes following childbirth the perineum is restored, but the anal sphincter is not united and incontinence results.

*Symptoms.*—In from four to six weeks following the sphincter being severed it fully retracts, although to a variable degree. Sometimes the muscle ends are still bound together and the tear only enlarges the circle slightly. In other instances the ends are widely separated and the muscle, which is thick and short, makes but a shallow arc behind the anus, with a deep dimple in the skin of the posterior raphe behind the location of the muscle. A smooth glazed depression of the skin or sometimes puckered pits mark the location of the ends of the sphincter. Thus the ability of the patient to retain feces is not an index of the depth of the rent in the muscle, but rather of the extent of separation of the ends of the fibres. If the cicatrix binds the ends firmly together an efficient sphincter may still obtain. This approximation is what we hope to attain by a plastic operation in the less fortunate individuals who apply for help. The individual can usually retain formed feces satisfactorily, but as soon as the discharges are at all liquid, incontinence occurs. The result is that the patient limits his diet and uses drugs to maintain a degree of constipation which soon interferes with proper alimentation. The unpleasant odor forces him from society and he soon becomes melancholy, thus adding another chain of complaints. If the mucosa pro-

lapses a rectal tenesmus appears. Many of these patients develop a nervous diarrhoea excited by the least rumbling of intestinal gases. The whole complex of symptoms tends to reduce the patient's physical well being.

*Treatment.*—When the incontinence is only partial much relief is obtained with galvanic electricity, hot fomentations and a careful regulation of diet. These should be tried before resorting to operative interference.

*Operative Treatment.*—Any plastic operation to be successful must be based on an anatomical and functional knowledge of the structures involved, and knowing this the foremost fundamental idea and the only rational aim of the surgeon in all such plastic work should be to restore the damaged parts to as near as possible their normal functional state, or, in other words, to follow nature's plan in the reformation of the region under his consideration. The relief of incontinence depends upon our ability to reunite the severed ends of the sphincter. Our success also depends upon the amount of muscle destroyed in the original operation or the subsequent sloughing of tissues and also upon the length of time elapsed since the damage was sustained. The muscle cut and therefore unused will atrophy and in time is useless if reunited, or it may have degenerated so much that it cannot be found. If much tissue has been destroyed the muscle fibres may be too short to be brought together. Usually, however, the fibres, because of their imperfect position, are too long to act and the operation shortens and brings the ends into normal position.

If the patient suffers with a diarrhoea the colon will need a thorough cleaning out a few days before the operation. It is well to give a light purge twenty-four hours before the time set for operation, so that its effect will be spent during the next twelve hours. Three hours before operation the colon is to be freed of all fecal matter by an enema given until it returns clear.

CASE REPORT A-36.—Had what appears from the history to have been an abscessed Bartholin gland, although she says she "had two abscesses of the rectum, one of which opened spontaneously and the other was lanced." Fistulæ resulted which were operated upon in November and again in February (eighteen and fourteen months ago, respectively). Since the last operation there has been a protrusion at

the anus but no bleeding. There is a sense of pressure in the pelvis and an almost constant desire to go to stool.

On examination there is found a narrow scar in the left anterior part of the anus extending outward and anteriorly about an inch. Also on the right side of the anus there is an irregular scar about one-half an inch wide and extending laterally about an inch. This latter is an irregular scar. The anal mucosa protrudes and the anus gapes open about one-half inch.

On digital examination the contractile bite of the sphincter is missing. With one finger within the rectum and the thumb palpating the anal rim the sphincter can be felt around the posterior half of the anus and is found to contract under the patient's voluntary effort.

Fig. 1 is a photograph of the patient when she came for examination. It will be seen that the radial folds of the skin extend outward posteriorly from the anus only. The anal mucosa prolapses on the anterior and right walls. The relaxation of the perineum allows the vaginal wall to prolapse. This patient has never borne a child.

*Operation.*—The patient was placed in the dorsal position and a horseshoe-shaped incision was made through the skin around the anterior commissure of the anus beginning at or back of the dimple marking the sphincter end on her right side and extending past the sphincteric dimple on the left side and a flap of tissue was dissected out and the ends of the sphincter muscle exposed, being careful to remove only so much of the muscle as was absolutely necessary, because that muscle was already very short. In a case of long standing the sphincter ends may be indistinct. If so, the muscle may be pinched or pulled upon to excite contraction, and the ends thereby discovered or the muscle may be rolled between the operator's thumb and finger. If the sphincter ends have been widely separated an extensive exposure of the tissues will be necessary. In this patient the sphincter had been cut in two places and the whole anterior portion of the muscle had degenerated.

The sphincter ends having been found were now freed from the surrounding tissues and drawn out with clamps until they could be well approximated and were fastened together with a mattress suture of No. 1 chromic catgut. A single suture was also fastened through the muscle at its sides to approximate a wide surface. A tension

**FIG. 1.**



Incontinent anal sphincter.

Note puckering of the skin in the posterior quadrant but a  
laxness of the anterior portion.

**FIG. 2.**



Shows line of incision to expose the muscle  
tissue.

FIG. 3.



Ends of the sphincter picked up and the initial sutures in place.

FIG. 4.



A good broad surface of the muscle is firmly approximated.

FIG. 5.



Anchor stitch is placed before the wound is closed.

FIG. 6.



Skin sutures are in place. The adhesive strap across the buttocks is not shown.

suture of paraffined silk was now passed through the skin behind the ends of the incision transfixing the sphincter muscle on that side, passed up through the perineal body and then out, transfixing the muscle on the opposite side of the wound. As this suture is fastened it reinforces the sutures in the divided muscle ends and prevents tension by the involuntary contractions of the sphincter.

After all oozing was checked the skin sutures of fine paraffined silk were placed to close the wound and then the tension suture was tied tight enough over a gauze pad to relieve tension on the buried sutures approximating the sphincter ends. The buttocks were strapped together with a broad band of adhesive plaster to prevent accidental traction on the wound. Further protection was assured by binding the patient's knees together for the first few hours until she had regained control of herself after the anæsthetic.

When the operation is well performed and is successful in bringing direct opposition of the muscle ends, the skin folds radiate from the anus and form a complete star all around it just as they did before the sphincter was injured.

The patient's bowels were confined eight days by allowing as food only egg albumen, two tablespoonfuls in water every three hours. On the eighth day the patient received an enema of water 6 ounces and mineral oil 3 ounces. This enema was repeated daily thereafter until all hard fecal masses in the colon were removed. No laxative was given. Our patient turned on her side or face to urinate and sometimes it is best to have the patient lie on her side when the bowels move to avoid straining. When albumen alone is given there are very few scybala and the evacuation is without difficulty or danger.

In every case the wound must be carefully examined daily to be sure no infection or abscess is developing. Should an abscess occur it must be incised early and drained in such a direction, if possible, that the united ends of the sphincter will not be injured.

The patient should remain in bed for two weeks after the operation and for four weeks more should go about with care, avoiding hard work or lifting.

If the sphincter has been divided in more than one place and the separated section still has contractile power, the repair operation should be performed in several steps, repairing only one injury at each

sitting. The second operation may be attempted a month after the first.

Narrowing of the anal outlet will greatly benefit the patient when the muscle function cannot be restored by plastic operation. In such a case a buried kangaroo tendon is passed around the anus at the level of the upper border of the external sphincter and tied firmly upon the index finger introduced into the anus. If these procedures fail or cannot be used, a left-sided colostomy with an abdominal anus should be advised.

30 NORTH MICHIGAN AVENUE.

## IRREDUCIBLE UTERINE INVERSION AND ITS SURGICAL TREATMENT

By D. J. BONTE, M.D.

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WHEN the physician is called upon to treat a case of inversion of the uterus, his first care should be to attempt manual reduction, exactly as he would resort to taxis in the case of a strangulated hernia. This procedure—the most rational and logical—is almost always successful in instances of recent inversion and occasionally spontaneous disinvagination occurs because involution of the uterus will bring about a normal state of affairs after the lapse of a few days.

But when one is dealing with a chronic inversion of the uterus, when the organ has undergone involution, the problem is more difficult to solve. The inversion may be so irreducible that even with the uterus in hand post-mortem or after hysterectomy, it cannot be reduced, and it can readily be surmised that in these cases which unfortunately still are far from infrequent, the surgeon has been obliged to resort to hysterectomy. Let us just consider the conditions which prevent replacing the uterus in its normal position.

One of the principal factors of irreducible inversion is spasm or contraction of the uterine muscle. This spasm may only involve the cervix, in which case a series of muscular rings, forming a true sphincter, will be found a few centimetres from the external os. The muscle fibres forming this ring may contract and thus prevent reduction. This obstacle is easily felt when reduction of the inversion is essayed by the procedure of central taxis, the fingers united so as to form a cone exercise a pressure on the inverted fundus uteri and produce a slight infundibulum, but when an attempt is made to drive them deeper they are arrested at the cervix and in some cases the resistance cannot be overcome with any degree of safety.

This contraction may even occur without any attempt at reduction being made, and cases have been recorded in which true intestinal strangulation has occurred. In a medico-legal autopsy performed on a female post-partum, when the abdomen was opened the uterus was



found depressed into a deep cul-de-sac measuring fifteen centimetres deep and ten centimetres in breadth, into which a loop of intestine, thirty centimetres in length, had become engaged, and being strongly compressed by the cervix offered a blackish-red tint and was with great difficulty disengaged.

In other cases the muscular contraction or spasm is not limited to the cervix; it involves the uterine muscle, and this condition may exist even when the lesion is of long standing. In a case of chronic inversion dating back sixteen months, the surgeon attempted manual reduction, but at each essay the uterus became hard and rounded and could not be depressed by the fingers. It was only after several tentatives and under deep anæsthesia that the organ became relaxed and could be reduced.

When the inversion is of long standing, very marked changes arise in the structures of the uterus, the muscle fibres decrease in number, fibrous tissue invades the walls of the organ and after contraction, retraction ensues which renders the inversion absolutely irreducible.

In these circumstances, it will be noted that the entrance of the peritoneal orifice of the uterus is small and retracted—almost filiform. It is puckered like the opening of a string purse and usually the corresponding portion of the corpus uteri is strangulated and the inverted uterus looks like a polypus with a rather small pedicle.

The older writers—Barnes and Gaillard Thomas—looked upon this condition as the principal obstacle to reduction, and in order to facilitate this procedure they advised first doing a dilatation of the cervix. Barnes even made longitudinal incisions in the muscular rings of the organ, while the American surgeon did not hesitate to open the peritoneal cavity and with a special forceps directly dilated the strictured ring, a bold procedure in his day.

As with the cervix, the corpus uteri can become very hard, even scirrhus, in which case it becomes retracted and sensibly decreases in size. There is at the museum of St. Bartholomew's Hospital a specimen of an inverted uterus which had become so small that the orifice of the pocket that it formed was not larger than the calibre of a duck's quill.

Retraction of the cervix is not only a direct obstacle to reduction of the uterus into the peritoneal cavity; it also acts indirectly by compressing the vessels which enter the fundus uteri, thus producing con-

gestion and œdema of the organ. Therefore, in these circumstances it can readily be surmised that the uterus is impeded in its physiological retraction and requires more time to contract than in ordinary puerperal involution.

In these cases one will be dealing with an elastic, globular tumor which has made its exit from the vagina and hangs between the thighs. Its surface is greater at the base than at the apex, where it is strangulated by the uterine orifice. This tumefaction remains manifest for a long time, so much so that it has been known to be the size of a pear at the end of from three to seven months after the labor. Given such a considerable mass, all attempts at reduction should be preceded by massage until the uterine congestion has been overcome, but taxis in these cases must be accomplished with the closed fist and not with the fingers brought together to form a wedge.

Strictly speaking, mobility of the uterus is not a cause of irreducibility of an inversion, but very frequently it greatly complicates the manoeuvres of reduction and renders them extremely difficult. It is especially in the procedure by taxis that it will cause difficulty; if in these circumstances direct pressure is brought to bear on the fundus of the uterus, if the operator has not taken the precaution of fixing the cervix with tooth-forceps, very serious tears of the vagina and deep structures will inevitably ensue, and White, of Buffalo, has put on record an example of this unfortunate occurrence.

In order to abviate this mobility, a very great number of procedures have been devised and recommended. Some surgeons fix the uterus by exercising a counter-pressure on the cervix through the abdominal walls, but this method is bad because it prevents the fundus of the uterine globe from being pushed back into the abdominal cavity. It is likewise often without effect because the organ slides and continually changes its position.

Other operators introduce two fingers within the rectum and hook them over the broad ligaments while they exercise pressure on the fundus of the uterus. This procedure often causes tears and laceration of the rectal mucosa and is otherwise not to be commended.

Very often a veritable localized inflammatory process of the peritoneum covering the infundibulum occurs. The peritoneal tugging, the difficulties brought to bear on the blood-supply of the organ from contraction of the cervix, provoke a congestion of the peritoneal serosa

and very soon serum will be found to transude from the dilated capillaries, the fibrin coagulates and a vascular neoformation with the production of numerous adhesions results.

When the uterine sac does not contain any viscera within it and communicates with the abdominal cavity by a narrow orifice, the walls come together and an inflammatory process has as a single result to bring about a complete occlusion of the infundibulum. At the same time, the round and utero-sacral ligaments become adherent to each other and form an inextensible cord which, at the point of exit from the obliterated sac, will spread over the pelvis.

The lesions may be still more complex; the inverted uterus can not right itself, the cervix may be dilated, and between the rectum and bladder a pocket exists which will contain either the ovaries, intestines, omentum, or even the bladder or rectum. All these viscera by the pressure they exercise upon each other, produce, after a time, adhesions between themselves and the peritoneum lining the sac. Many remarkable instances have been recorded in the past, before the days of antiseptic surgery, and even of late years deaths from this cause have been reported, when reduction of the inversion by taxis has been attempted.

On the other hand, the peritonitis of the infundibulum may be a fortunate occurrence in cases where the fundus uteri undergoes a necrobiosis from constriction and is eliminated, as the adherent serosa walls off the peritoneal cavity and thus diminishes the risks of infection.

Finally, to complete the list of the causes of irreducible inversion, I would refer to a curious complication of this condition, a serious complication at that, since it may require a treatment quite as radical as that of irreducible inversion—I refer to what we call in France an "*incoercible uterus*." In certain cases the inverted uterus loses all contractility, both of the cervix and corpus, and offers itself in the form of a soft, depressible tumor. Reduction is readily accomplished by simple taxis, but this is only temporary because the organ being deprived of any resistance, held by relaxed ligaments, becomes inverted again as soon as the pressure of taxis is relaxed.

It is an impossibility to restore the contractility of the uterus and all the means resorted to, such as counter-irritants, preparations of ergot and hot irrigations remain powerless. Baldy reported an

interesting case in which even ventrofixation did not prevent the recurrence of the inversion.

The accidents which may arise in irreducible uterine inversion are sufficiently serious to justify surgical interference, especially in young women. Not only is the condition intolerable for the patient, but it may give rise to very serious complications compromising the life of the woman.

In the first place there is pain of variable intensity, which usually decreases as the condition becomes chronic. But then there will be a constant painful sensation in the lower abdomen due to dragging on the peritoneum by the inverted organ.

The uterus being no longer held in place by its supporting ligaments, has a tendency to prolapse through the vagina and at the same time exercises traction on the peritoneum, round ligaments, tubes and ovaries. The patient instinctively tries to overcome the pain by remaining in the recumbent position and scarcely dares to leave her bed.

When the ovaries are within the infundibulum a special pain—similar to that resulting from pressure of the testicle—occurs, especially when the ovaries become inflamed or simply the seat of congestion at the time of the menses. The resistance of the uterine muscle prevents the congestive tumefaction of menstruation taking place, so that ovarian pains in these circumstances may be considerable.

Besides pain, uterine inversion often produces disturbances of micturition or defecation. Incontinence of urine results from irritation of the bladder, but usually there is a more or less complete retention. To void the urine, some patients are obliged to push back the tumor which fills the vagina and compresses the organs in the small pelvis, while in other cases the catheter must be used.

When inversion is complete and forms a tumor outside the vagina, very serious lesions may result. These are produced by friction from the clothing or from contact with irritating or septic fluids, such as the urine or vaginal secretions; the congestion and œdema of the uterus resulting from contraction of the cervix are also important complications. Ulcerative processes occur on the uterine mucosa, giving rise to considerable purulent discharge. These ulcerations have been known to undergo malignant transformation, a fact that must not be lost sight of.

The irritative lesions do not always remain limited to the uterine mucosa, as they may extend by continuity or by way of the lymphatics to neighboring structures and organs. Thus salpingitis, perimetritis or even generalized peritonitis have been met with in inversion.

In very infrequent cases, the inflammation extends to the vagina and vulva, and when the labia majora became tumefied, they may cause strangulation of the uterus.

Hemorrhage is yet another important complication and a very frequent one. The uterine structures are, in the normal state, among the most vascular of the body, the special arrangement of its helicine arteries and sinuses demonstrates this fact. Now, when inversion has taken place, contraction of the cervix produces a kind of stasis which gorges the organ with blood and this congestion attains its maximum at the time of the menses, the result being the occurrence of very severe hemorrhage. The slightest contact, such as vaginal examination, is sufficient to set up considerable loss of blood.

On account of these hemorrhages the patients are pale, very anæmic and subject to dizziness or syncope, and when the metrorrhagia is frequent the organism has not the time to repair the loss of blood, so that the latter is replaced by a serous discharge hardly tinged with blood. When a patient is allowed to reach this condition, the prognosis is very serious.

In some cases, particularly in elderly women, all these symptoms may decrease in intensity or even disappear. The uterine mucosa becomes hard and fibrous and insensible to irritating agents, while the hemorrhages may cease, especially when menstruation stops either from the menopause or nursing.

In these circumstances the inversion may be perfectly compatible with life and will give rise to few accidents.

Finally, to complete the subject of prognosis of chronic inversion of the uterus, spontaneous reduction of the inversion should be mentioned. In several instances this has occurred after vain attempts at reduction by taxis. The cause of these spontaneous reductions are variable. In one case reported by Darvan, it was due to an enormous accumulation of feces in the rectum, which filled the concavity of the sacrum, pushing the uterus towards the pubis, thus acting like a pessary.

In another case reduction was obtained by simply keeping the

patient in bed. In all these instances of spontaneous reduction the suspensory apparatus of the uterus plays an important part, the accumulation of *fæces* in the rectum, repeated attempts at coitus, the horizontal position and traumatism of the perineum or pelvis have the effect of pushing up the uterus. The ligaments—especially the round ligaments—from their tonicity and tendency to retract, tend to produce disinvagination of the uterus because they are inserted on the circumference of the pelvis and also on the fundus of the inverted organ.

Other ways of spontaneous cure have been recorded, but with loss of the organ from gangrene resulting from constriction of the cervix. The fundus undergoes necrobiosis and is eliminated, thus producing a hysterectomy by the hand of Nature, a piece of work which is not to be desired. Such instances were not uncommon before the advent of modern surgery and they not infrequently ended in a fatal septicæmia.

As to treatment of chronic uterine inversion, it is always operative if taxis fails. In what is to follow, I shall only refer to the operative treatment, as the technic of taxis by modern procedures is well known to all, but I would say here that hysterectomy is only indicated in cases of gangrene of the fundus or if malignant changes should have arisen at the site of ulcerative processes on the uterine mucosa.

The first operative procedure to be considered is that devised by Küstner, which is a combination of incision of the cervix as advocated by Barnes and fixing the cervix as recommended by Courty. It can be resorted to in a large number of cases which are irreducible. The technic is as follows:

A free incision is made in Douglas' pouch and by this opening a finger is introduced within the constricting ring and at the same time breaking down any existing peritoneal adhesions. Next, a longitudinal median incision of the posterior wall of the inverted uterus is made, beginning two centimetres from the fundus and carried to within two centimetres of the external os. It should include the entire structure with the peritoneum.

Reduction of the uterus is now accomplished, the index finger fixing the cervix through Douglas' pouch and the thumb of the same

hand pushing back the fundus. Suture of the uterine incision in two layers. Drainage through Douglas' cul-de-sac.

This procedure appears to me rational and it has unquestionably given brilliant results. It has the advantage of leaving the external os intact, while the second advantage of the procedure is the possibility of directly fixing the cervix through the incision in the posterior vaginal cul-de-sac, so that pressure on the fundus uteri will be more efficacious, but there are cases of chronic inversion which cannot be reduced by Küstner's procedure and then posterior colporhysterotomy, according to Duret's technic, must be done.

This operation possesses the immense advantage in that it will reduce any irreducible inversion and, therefore, does away with hysterectomy. The success of this operation appears to be due to two factors. The incision, including the entire length of the posterior wall of the uterus, removes all obstacles to reduction, whether they be seated in the cervix, fundus or infundibulum. Another advantage is that by the extensive incision of Douglas' pouch, the adhesions existing between the neighboring organs in case of peritonitis at the infundibulum can be broken down and the organs freed.

The second factor of success is to be found in the fact that reduction can be accomplished outside the vagina and a glance at Plate III shows that the operator can execute all the movements that he may desire, a simple vaginal retractor giving him a vast field of operation.

Reduction is, for that matter, as simple as possible. In Küstner's procedure the incision does not include the cervix, as it commences at the isthmus and, consequently, in order to reduce the fundus within the abdominal cavity and cause it to pass through the narrowed cervix, considerable pressure must be made, as well as to reduce the volume of the organ as far as possible. With the technic of colporhysterotomy that I am about to describe, disinvagination in the strict sense of the word is not done, the uterus is simply unrolled, so to speak, the cut surface being brought over on the front.

Another disadvantage of Küstner's procedure is the impossibility to suture the button-hole made in the uterine wall, so that peritoneal complications or uterine rupture with the next pregnancy may occur. In posterior colporhysterotomy reduction is accomplished in the vagina, and the uterine incision can be sutured under the direct

guidance of the eye before replacing the uterus within the peritoneal cavity.

The contra-indications for posterior colpohysterotomy are few and all reside in the condition of the uterus. When the inversion is complicated by fibromata, cancer, gangrenous metritis, pyometritis or serious lesions of the adnexa, no hesitation is possible, because the concomitant affection is, *per se*, a sufficient contra-indication. In elderly women, especially if the inversion is of long standing, hysterectomy may be preferable, because posterior colpohysterotomy would be more difficult to carry out, and if menopause has taken place it is unnecessary to preserve the uterus.

I prefer Duret's technic, which I shall now describe in detail. The preparation of the patient and field of operation will be attended to according to the preference of the surgeon, and I have nothing particular to offer in this respect.

The first step is the *incision of the posterior vaginal cul-de-sac*. The fundus uteri is seized with strong-teethed hysterectomy forceps and drawn downward and forward (see Plate I). By this traction the cervix becomes completely inverted and presents itself on its mucous aspect, readily made evident by the ramifications of the *arbor vitæ*. The pedicle of the tumor is grasped between the thumb and fingers and the cervix is located through the thickness of the tissues. A transversal incision *just above* this point is then made in the posterior vaginal cul-de-sac; the incision should be from three to four centimetres in length and prudently made, because the pelvic infundibulum may contain organs that must not be injured.

The second step is the *posterior vertical median incision of the uterus* (see Plate II). Through the posterior vaginal incision the uterine infundibulum is explored with the finger to ascertain whether or not it contains any viscera—intestine, ovary, etc. If adhesions exist they are separated, while the omentum or intestinal coils are kept out of the field of operation by proper packing. With the finger introduced into the infundibulum a vertical median incision is made comprising the entire thickness of the posterior wall of the uterus.

The incision is begun at the middle of the anterior border of the vaginal incision and ends at the fundus. It should be absolutely median, and it is only upon this condition that hemorrhage will be



avoided. When this has been made, the operator will have the cavity of the infundibulum—which represents the peritoneal surface of the inverted uterus—directly under the control of the sight.

The third step is *the reduction of the inversion* (see Plate III). Both thumbs are placed on the anterior (mucous surface) of the uterus, and with the other fingers the borders of the posterior incision are spread apart and finally the two halves of the uterus are turned back, so that the mucous membrane of the uterus which was outside now returns to form the normal uterine cavity. In other words, the incision which was posterior now becomes anterior.

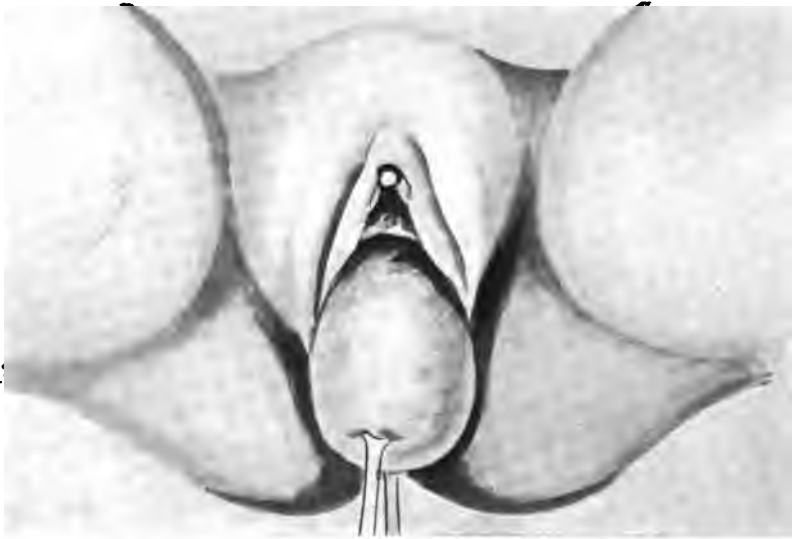
The fourth step is *suture of the corpus uteri as far as the isthmus* (see Plate IV). The uterine incision is sutured by commencing at the fundus, and is carried down to the isthmus. Two layers of suture are used, a deep muco-mucous and an upper layer including the muscle and peritoneum, both being made with fairly good-sized catgut. This suture is easy, because by drawing on the uterus with the forceps the incision can be brought outside the vagina.

The fifth step is *the reposition of the uterus* (see Plate V). The uterus now offering its peritoneal aspect is still in the vagina, and it must now be replaced into the peritoneal cavity through the posterior vaginal incision, which for this purpose is enlarged by making a second median incision, starting at the posterior border of the transverse incision and carried perpendicularly towards the vulva. Reposition of the uterus is then easily accomplished through this large opening, but the edges of the incision must be held by forceps during the manœuvre.

The sixth step is *suture of the cervix* (see Plate VI). After the uterus has been replaced within the abdomen, the operation is completed by suture of the cervix, which is still open on its posterior aspect. The anterior lip is caught and brought down, and a suture is made from the isthmus to the end of the cervix, using two layers of catgut.

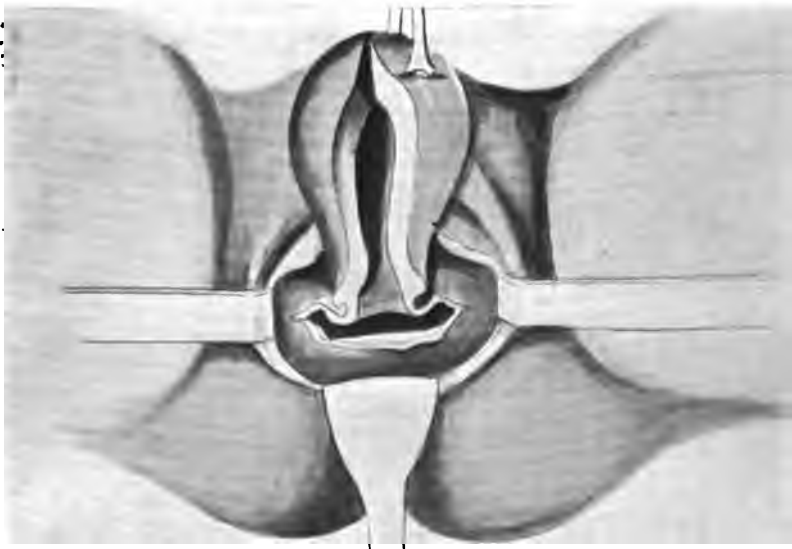
After this the posterior vaginal incision is packed with gauze for drainage and also for pushing the fundus forward and upward, thus overcoming any tendency to retroversion.

PLATE I.



Total uterine inversion. The uterus is drawn downward by forceps.

PLATE II.



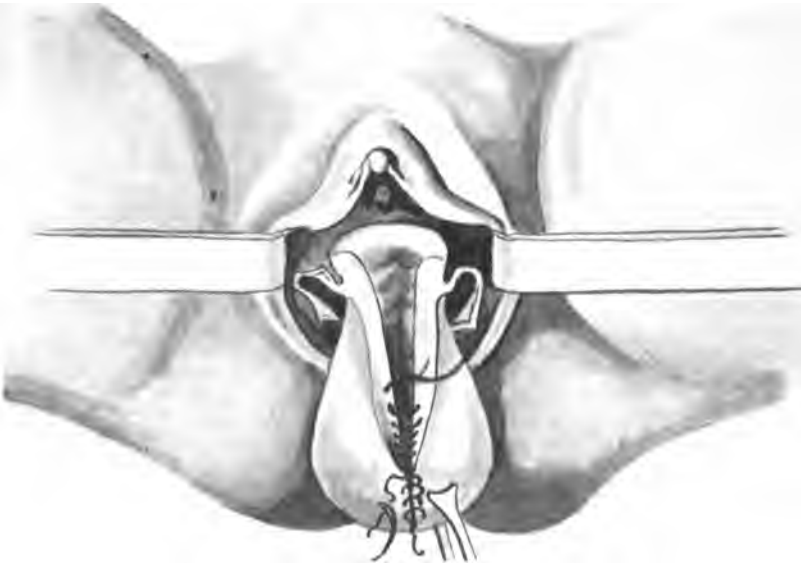
First and second steps of posterior hysterotomy. The uterus is raised upwards on the stretch. The posterior vaginal cul-de-sac is incised transversally. The inverted posterior wall of the uterus has been completely divided by a longitudinal incision which exposes the serous aspect of the organ.

PLATE III.



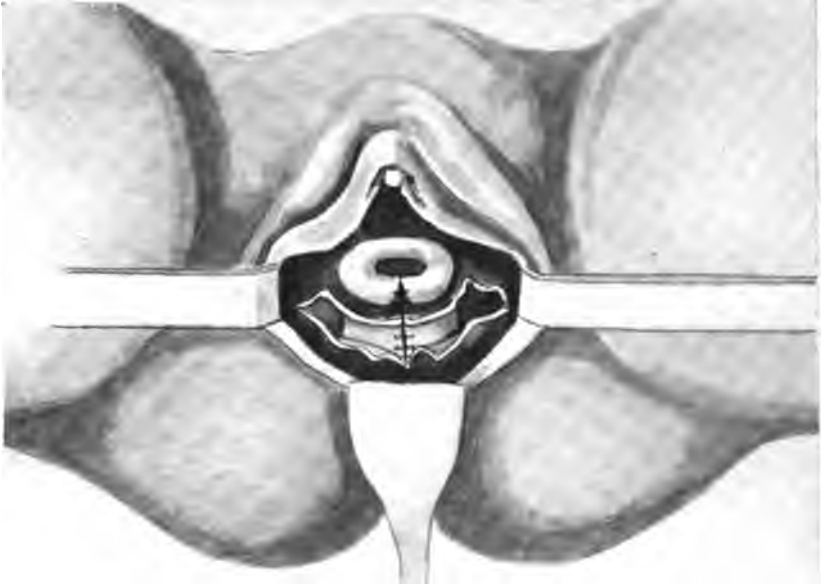
Third operative step. The operator is about to turn back the uterus so that the peritoneal surface of the organ becomes external.

PLATE IV.



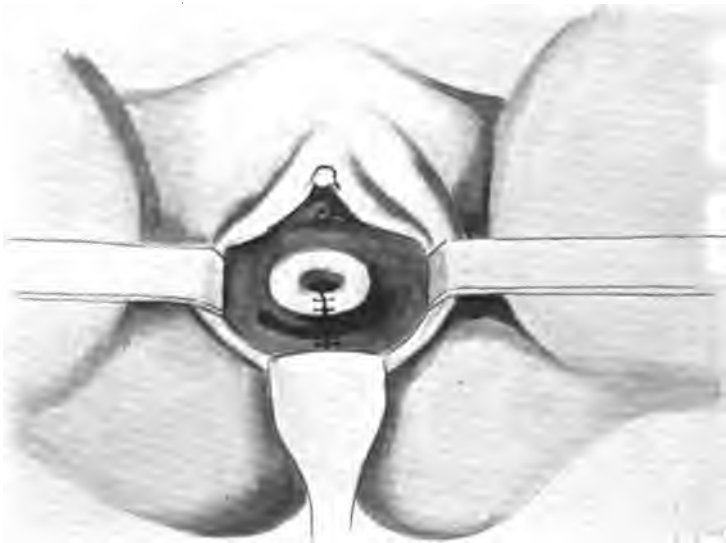
The uterus is now drawn downwards. The inversion has been reduced, the peritoneal surface is outside. The uterine incision is being closed by two layers of sutures, muco-mucous and sero-serous.

PLATE V.



Suture of the uterus having been made, the organ has been returned into the abdominal cavity through the vaginal incision which has been enlarged by a longitudinal incision. The cervix is sutured after the uterus has been returned into the abdomen.

PLATE VI.



Cervix sutured. The incision in the posterior cul-de sac is left open for gauze packing in order to avoid retroversion and for draining the abdominal cavity.



## THE TREATMENT OF COMPLICATED FRACTURES \*

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SOLUTIONS of osseous continuity, regardless of their anatomic situation and manner of production, may be divided into two general groups, viz., simple and complicated; and the latter group is susceptible of subdivision into numerous varieties and types. The scope of this paper precludes detail consideration of simple types, and time will permit only a general outline of the treatment of complicated fractures.

As a general proposition, it may be stated that any fracture which requires operative intervention to secure reduction and maintenance of fragments or to promote union may be classified as complicated; and that in the majority of complicated fractures the open method of treatment must be employed to insure correct anatomic apposition and restoration of function.

At the outset it must be remembered that, regardless of how simple a fracture may appear from the manner of its causation, the surgeon who fails to carefully examine his patient by modern methods to confirm the clinical diagnosis and to determine the presence or absence of complications, not only neglects his duty but is courting disaster by subjecting himself to the liability of suit for malpractice provided an unfavorable outcome ensues. The most valuable agency yet devised for the diagnosis of fractures of every type and the prevention of damage suits is the Röntgen-ray, and the importance of having two or more plates made at different angles both before and after reduction cannot be too strongly emphasized. The surgeon, in justice to himself and his patient, should insist upon having an X-ray examination made in every fracture, and with the present multiplicity of X-ray units and competent laboratory workers there is little excuse for failure to do so.

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\* Portions of this paper appeared in the *American Journal of Surgery*, March, 1920.

For obvious reasons the treatment of complicated fractures seems impossible of complete technical standardization, in that particular differing from many other surgical lesions; and while there are certain features upon which practical agreement has been reached, for the most part treatment must remain individualized. However, many improvements have been made within the last decade, and the main object of this paper is to direct attention to some of the most important with especial reference to the open method.

Not many years ago it was considered extremely hazardous to incise the site of an apparently simple fracture—i.e., to convert a simple into a compound fracture—even where it was evident the fragments were imperfectly apposed; but the surgeon of to-day who does not resort to open operation under such circumstances is subject to criticism. There can be no question about the necessity of open operation in complicated fracture; and this applies to simple fracture where the fragments cannot be maintained in proper anatomic apposition by the closed method. While due credit for improvement in aseptic methods and perfection in operative technic of bone surgery must be accorded Mr. Lane, of London, the valuable work accomplished by Geiger, Albee, Hibbs, Murphy and many other surgeons must not be overlooked.

Indiscriminate operations on simple fractures, where good anatomic apposition and restoration of function can be secured by the closed method, has been rightly condemned. However, over-riding of the fragments, displacement, shortening or malposition with resulting deformity, should be corrected by the open method. The time has long since passed when good results and satisfactory function can be claimed if there is over-riding of fragments, shortening or deformity of the limb following the reduction of fractures. While a limited amount of motion and function may be secured in a deformed limb, one must consider disability arising from displaced bone, shortening or malposition, also the effect on blood-vessels, nerves or a joint, which may become manifest after the patient has been dismissed. In many instances varicose veins, neuritis, "strained" joint, "rheumatism," and other types of disability due to bony malposition following fracture could have been prevented by open operation.

In my opinion, the open method is indicated in the following types of fracture regardless of their anatomic situation:

1. Where there is considerable displacement of fragments which cannot be otherwise corrected;
2. Where complete reduction and maintenance cannot be secured under manipulation by the closed method;
3. Where manipulation causes undue trauma to adjacent soft structures, blood-vessels, nerves, etc.;
4. Where spiculae of bone or soft tissues are interposed between fragments, thus preventing union;
5. Where the fragments are rotated upon each other and cannot be maintained in proper position;
6. In spiral, multiple and certain oblique fractures where perfect anatomic apposition cannot be maintained;
7. In every fracture entailing serious damage to either nerves or blood-vessels;
8. In ununited fractures and in so-called "vicious union;"
9. In fractures complicated by dislocation or in close proximity to joints including intra-articular lesions;
10. In all femoral fractures which cannot otherwise be maintained in perfect anatomic apposition;
11. In all compound fractures in any situation.

In the foregoing types experience has demonstrated that more favorable ultimate results may be obtained by open operation with internal fixation than by attempting reduction and maintenance of fragments by manipulation and external fixation, provided, of course, the surgeon is equipped with the necessary modern instruments and his surroundings are such that perfect asepsis can be maintained. In intra-articular fractures the operative technic is particularly difficult and should be undertaken only by those who have had considerable experience in bone work.

My views are in accord with those of Geiger, who says (quoting Hixrot) that the most striking contraindications for operation on broken bones are inexperience on the part of the surgeon, unsuitable surroundings, and insufficient equipment; furthermore, the operator to be successful must have intimate knowledge of the anatomy of the region to be operated upon, also the action and function of muscles and ligaments involved in the injured area. The reduction of fractures should invariably be checked by Röntgen-ray examination to determine whether or not the fragments are in perfect apposition.



However, the observation is noteworthy that the X-ray often seems to exaggerate the deformity if one is unfamiliar with the angle at which the picture was made.

In answer to the question frequently asked: "Why is open operation necessary?" the following pertinent facts seem worthy of emphasis:

1. The greater percentage of instances in which satisfactory functional results are secured is in favor of open operation; in special fractures, such as both bones of the forearm and the femur, open operation should always be performed if the position is faulty; furthermore, the results are more favorable from open operation as age advances.

2. Perfect anatomic apposition is secured, hence better cosmetic results; "and to be certain of good function, be sure of correct anatomic apposition."

3. Union is earlier following the open method, *i.e.*, where bone grafts are used; but may be prolonged when foreign material is employed.

Where necessary in non-infected fractures operation is indicated early, *i.e.*, if possible within five or six hours after injury. This is regarded as the most favorable time, provided undue trauma has not been inflicted by attempted reduction under the closed method. Examination by the Röntgen-ray is essential to determine the type of fracture, the position of the fragments and the advisability of operation before the tissues have been traumatized. If the patient is seen after twenty-four hours and there is evidence of considerable damage to adjacent soft parts, produced either by the initial injury or attempted reduction, operation should be delayed ten or fifteen days, or until the tissues have assumed a more normal condition.

The importance of strict asepsis as one of the essential fundamentals in successful bone grafting cannot be too frequently reiterated. Unless there are legitimate contraindications autogenous grafts and metal fixation methods are to be preferred. In non-infected cases, where the anatomy of the injured part requires no unusual strain at the fracture site, the bone graft should be employed. Personally, I prefer the inlay or sliding graft to the intermedullary where conditions will permit

Certain mechanical and physiological principles must govern the procedure regardless of the material employed, *i.e.*, whether the intermedullary, a transplant from the tibia, the rib, or a sliding graft. In making his incision the surgeon should constantly remember the anatomy of important structures involved; proceed gently and accurately with the least possible trauma; after reaching the bone at the fracture site careful examination with proper instruments (not with the hands) will determine whether an inlay, a medullary or a sliding graft is most appropriate. If an inlay or sliding graft be used, it should be cut to fit snugly into the groove made to receive it; the graft should include periosteum, compact bone and endosteum, and be placed so that intimate contact is secured between periosteum and periosteum, compact bone and compact bone, endosteum and endosteum, and be retained by autogenous bone pegs or kangaroo tendon to insure perfect apposition.

Regardless of what may be the opinion expressed by others, in cutting the graft I prefer a single saw; if a "twin-saw" be used the graft will be smaller than the groove made to receive it, and thus improper apposition and imperfect union may be expected. It is important that the graft shall not fit too tightly, otherwise pressure may produce bone-cell necrosis and thus retard union. In cutting the graft the speed of the motor should be such that the heat will not destroy the bone cells; water flowing over the saw to cool it is inadvisable as this washes away material which should remain in contact with the bone.

If an intermedullary graft or dowel be used, all periosteum must be removed from the portion which extends into the canal of the bone; but if there is loss of substance from the ends of the fragments, the periosteum should be left on the graft in that space. The dowel should be cut to fit snugly and all ivory-like bone removed from the canal so the dowel will come into intimate contact with healthy bone and marrow. Perfect hæmostasis must be maintained in this method as well as in the inlay; a blood clot may separate the soft structures from the bone and the graft will in consequence be inadequately nourished. This has been the cause of failure in not a few instances.

If a rib or section of bone from another portion of the body be used, similar principles must be observed, bearing in mind the essential element of a transplant is the cortical bone because of its resist-

ance to change, its limited vascular supply, its power to live on a minimum amount of nourishment, and its active osteogenetic properties which cause it to grow with rapidity and firmness.

Variation in technic and material may be required in special fractures. For example, bone pegs are used in fractures of the anatomical neck of the humerus and may be advantageous in certain other special situations.

It must be remembered that the purpose of fixation by bone grafts, pegs, etc., is to promote union and maintain the fragments in direct apposition and continuity—not for strength. Therefore, the part grafted must be handled carefully and the limb completely immobilized in a plaster cast, including one or more joints proximal and distal to the graft.

In the presence of infection metal plates should be used for internal fixation in preference to grafts, also where there is great strain on the bone at the fracture site and in certain of the larger bones when time is important; a bone can be plated much quicker than a graft can be inserted.

In plating fractures similar precautions must be exercised as to hæmostasis, asepsis and in handling the tissues, as in bone grafting. Make a free incision to the periosteum, but do not disturb it; if the periosteum has been torn or stripped from the bone, it should be replaced; no lacerated periosteum should be allowed to project into the soft structures, as it has osteogenetic power at the osteo-periosteal angle when osteoblasts are attached; hence, exuberant callus formation with traumatic exostosis may be expected as the logical result. This is serious when near a joint, as it will cause limitation of motion; it may involve important nerves and blood-vessels; callus is also troublesome when deposited in muscles, producing severe pain and limited motion unless absorbed or removed. This is important in bone grafting as well as in plating.

Before the plate is applied the ends of the bone must be freed of foreign material, such as blood clot, bony spiculæ and soft tissue; curette the canal until healthy bone marrow is reached. Be conservative of bone, i.e., preserve every portion where there is no loss of substance; jagged ends will become dovetailed and prevent shortening. Use a plate as long as the anatomic situation will permit and

omit the end screws nearest the fracture line; screws placed too close to the fracture will prolong and sometimes prevent union.

The plate having been applied, all vessels ligated and oozing controlled, close the soft structures carefully by the layer method, using absorbable sutures. Either alcohol or a dry dressing is then applied and the limb immobilized in a plaster cast. The operation should never be performed with a tourniquet applied, lest an overlooked vessel begin bleeding with the production of hæmatoma.

The wiring or nailing together of the fragments is indicated in the treatment of certain special fractures, but as the field for these measures is limited I shall not dwell on that phase of the subject.

It can no longer be doubted that plating of bones in the presence of infection has a legitimate place in surgery, although the indiscriminate plating of infected fractures is justly condemned. After plating a badly infected recent fracture—where the local circulation is greatly disturbed because insufficient time has elapsed for establishment of collateral circulation through the lacerated and bruised tissues and where vitality of the entire area is markedly lowered—one need anticipate nothing but failure.

In operating upon a compound, infected fracture, the original opening should be sufficiently enlarged to enable further work to be accomplished with ease. The operator should carefully remove all scar tissue, sequestra, callus and bone fragments whose viability is questionable, also all ivory-like bone from the ends of the fragments to be plated, thus leaving a bleeding area beyond the line of demarcation. Fibrous tissue must be removed from the canal until healthy bone marrow is reached.

In most instances the fragments will not fit closely enough to obstruct drainage, but if this should happen a drainage opening must be made with chisel. This is important, otherwise the development of osteomyelitis is likely.

The plate should be so applied as to not interfere with coaptation of the soft structures and the bone at the point where first union is expected. Neglect of this precaution may cause destruction of the bony fragments. Partially close the incision, covering as much of the bone as possible without obstructing drainage. In securing immobilization with a plaster cast a "spanner" is embedded in the

plaster to afford additional strength in the region through which daily dressings are to be made. The danger of herniation of the leg through the large opening in the cast with curvature at the fracture site may be obviated by the use of one or two ordinary forearm splints over the space after the dressings are applied.

The plate should be removed when the bones have sufficiently united to prevent displacement of the fragments. This can be done without an anæsthetic and without removing the cast. As a rule, within eight to ten weeks union will have become sufficiently solid to permit discontinuation of the plaster cast.

# Neurology

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## CRIMINAL ANTHROPOLOGY \*

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EVER since Cesare Lombroso published his monumental work, "L'Uomo Delinquente," the students of sociology and criminology have been waging constant warfare for and against the conclusions and deductions made in this famous volume. The question as to whether there exists a "criminal type" of man continues still to draw the heated fires of controversy. Criminal anthropology seems to have been constantly misunderstood because there has been a confusion between the "technical value of anthropological data concerning the criminal man and their scientific function in criminal sociology."

The real function of criminal anthropology is to determine whether the criminal is normal or abnormal; to learn the nature of his abnormalities and to ascertain whether or not they are of such a nature as to respond to treatment and correction.

The results and conclusions herein stated are from my daily observation and contact with all classes of criminals whose number totaled 5000, and these conclusions of necessity are empirical. These observations were made in the psychopathic laboratory at the Indiana State Prison and the Indiana Hospital for Insane Criminals at Michigan City, Indiana, where the author was formerly the Medical Superintendent.

### HEADS

It has been my observation that the heads of certain criminals when compared with persons living at liberty do not show a very marked difference in size; small heads and large heads have been observed with greater frequency among prisoners than have heads of medium size.

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\* Excerpts from lecture delivered to classes in Criminology at Summer School, University of California, Berkeley, California, July, 1920.

Asymmetry of the cranial vault is quite common, and I believe is more often seen in criminals than in an equal number of non-criminals. The cranial anomalies, such as plagiocephaly, macrocephaly, doliochocephaly, brachycephaly and acrocephaly are found among the insane, epileptic and feeble-minded persons in about the same ratio.

Certain anatomists have devoted much attention and study to the brains of criminals. They have carefully noted the weight, size and structural peculiarities, but however deserving their studies have been, they have shed but little light upon criminal anthropology.

The following brain weights indicate at least the negative value of this method of examination and comparison: "Oliver Cromwell's brain weighed 82.29 ounces; Lord Byron's, 79 ounces; Cuviers', 64 ounces; Ruloff's, a thief and murderer, 59 ounces; an adult idiot's, 54.95 ounces; Daniel Webster's, 53.50 ounces, and Gambetta's brain had the weight and size common to a microcephalic idiot."

#### EARS

I have noticed that deformed and anomalous ears are very common in prison. The Morel type of ear is very frequently observed, as is the presence of Darwinian tubercles. It has been noted that the Darwinian tubercle appears very often among the insane, epileptic and feeble-minded and probably occurs with the same frequency among prisoners. The negro criminals presented with great regularity the small shell ear, but this type seems to be characteristic of the American negro. Large ears are very common among convicts, both the lateral and the vertical measurements sharing in this excessive development. It was not unusual to find that the posterior branch of the division of the antihelix was subdivided into a Y-shaped extremity. This same phenomenon is common among certain types of monkeys and in individuals who have not obtained their full development. Among other abnormalities of the ear was found the absence of the helix and the antihelix, and malformations of the lobule which was often directly adhered to the side of the head without any indenture intervening between the lobe and its attachment. Very frequently the ears stood out from the head at a right angle and in nearly every instance of this kind the ears were very poorly developed. One is also impressed by the great number of deformed ears

which have been mutilated in fights. Frequently prisoners have had portions of their ears bitten entirely off by their antagonists in their physical encounters; this is especially true of negro criminals. The cauliflower ear owes its origin for the most part to traumatism, and not a few cases, I believe, are due to the too frequent, unnecessary, and brutal use of the police club. There is no criminal type of ear, but the criminals' ears on a whole belong to the degenerative types that are common to all classes of defectives.

#### EYES

Goring states after his study of English convicts:

"It will be seen \* \* \* that fraudulent offenders have rather better, and thieves have rather worse, eyesight than offenders generally. The amount of this association, however, between the eyesight of criminals at constant age and the nature of their crime is very small, and for all practical purposes may be regarded as negligible."

In the psychopathic laboratory at the Indiana State Prison nothing particularly striking about the eyes has been noted, except the great frequency of the arcus senilis and pupillary irregularities which indicate early degenerative morphological changes, usually the result of luetic infection. About 50 per cent. of the prisoners showed at the time of their entrance examination some defects of vision. These defects may generally be classified under three heads: congenital defect, such as myopia; astigmatism and hypermetropia; errors of refraction, due to acquired disease or traumatism and various degrees of presbyopia due to an advance in years.

#### NOSES

Ottolenghi devoted himself to the careful study and measurement of the criminal's nose. He even described the nose of the criminal in general to be of a certain kind, stating, as a rule, that it was rectilinear, of medium length with a broad base. He reports that the average thief's nose is of a rectilinear type, frequently curved with a depression backward and with the base of the nose upturned. He says that the sexual offender's nose is rectilinear, but of irregular profile and quite prominent. My observation of five thousand criminals has led me to the conclusion that there is no criminal type of nose, and this external olfactory organ varies in size and shape as it does



in ordinary civilian population. It was observed, however, that quite a noticeable percentage of prisoners had very defectively shaped noses. These deformities were due to traumatism, inflicted by fights, policemen's clubs and nasal syphilis. Doctor Stanley, of San Quentin prison, has noticed this increase in deformed noses from causes just mentioned; and he has devised a very ingenious method of surgical treatment to correct these nasal irregularities. The results of his surgical treatment are so perfect in many instances that the prisoners could not be identified by their original prison photographs.

#### HAIR

In common with certain other observers I have found anomalies of hair, there being a marked absence of a facial beard, especially upon the chin; often their skins were soft and delicate as a child's, and frequently not a few male prisoners above thirty years of age were able to go without shaving. Lack of hair upon the chest was very noticeable. These eunuchoid conditions of hirsute development were especially common in the prisoners guilty of homosexual crimes. Many of the feeble-minded prisoners exhibited excessive hairy development of the scalp, the hair line often coming within three-fourths of an inch of the eye-brows, which were very often continuous across the forehead and very heavy. Criminals who committed crimes of violence were often very hirsute, especially upon their lower limbs. It was not uncommon to meet with a prolific hair development upon the back.

Much has been written about the color of the hair, but so far the statistics furnished upon this subject are not conclusive. I have observed that gray hair was quite common among the prisoners of the Indiana State Prison, but I think this fact may be attributed to the age of the prisoners, to premature senility, and to the effects of syphilis. Only one case of baldness was present among two hundred insane criminals, and this condition was also exceedingly rare among the inmates of the prison.

#### CRIMINAL PHYSIOGNOMY

The criminal's physiognomy has been a matter of speculation for many years. People in general have regarded the face as an index to determine the good or evil in those they meet. They have come to

look upon the physiognomy as an animated reflection of the individual's mentality and morality. It is certain that the face mirrors the human hatreds, passions, and emotions. Ellis says, in speaking of criminal physiognomy: "There are, for instance, a large number of proverbs in which some of the most recent results reached by criminal anthropologists of to-day, were in long ages back crystallized by popular intelligence. Such are the Roman saying, 'Little beard and little colour; there is nothing worse under heaven;' the French, 'God preserve me from the beardless man;' The Tuscan, 'Salute from afar the beardless man and the bearded woman;' the Venetian, 'Trust not the woman with a man's voice.'"

It is a common practice for most individuals to form a favorable or unfavorable opinion of people they meet, and in many instances time proves that the first impression is correct. Of course signal mistakes are made by this manner of judging people. In defense of this universal practice of judging others Ellis remarks: "It is a mistake to attempt to stifle such instinctive impressions as irrational. They are part of the organized experiences of the race, and subject to intellectual control; they are legitimate guides to conduct."

Ferri, Garofalo, and Lombroso have told of instances where they were able to single out individuals who were murderers by inspection of the face and head. As a careful observer views prisoners in the dining-room of penal institutions or on the drill ground or in the prison chapel, he is struck by the fact that good looking faces are very rare.

A survey of the photographs to be found in any parole violators' record book will show that ugliness and facial distortion generally hold sway. Some ingenious individuals have attempted to offer a reason for this fact by saying that the prison uniform and the closely cropped hair were responsible for the ugliness, but this lame excuse does not hold good where the prisoner's photograph is taken in street dress and with his hair untouched by the barber. There are, of course, exceptions to all rules; very rarely a handsome prisoner is observed. Among the causes for the lack of good looks is the great frequency of prognathism or the reverse, high cheek bones, deformed noses, low foreheads, thick lips, cross eyes, and other facial irregularities; but these unfortunate conditions do not make an individual

a criminal as certain investigators once erroneously held. On the other hand, the individual who possesses to any great degree these evidences of anatomical degeneracy undoubtedly belong to an inferior physical type.

#### SKIN

It has been a very popular practice of criminologists to refer to the anæmic and cachectic appearance of prisoners. While such conditions do exist in those prisons which lack an abundance of fresh air and sunshine, this pallor of the skin cannot be described as one belonging exclusively to criminals, for it is also noted in those persons who are shut in from air and sunshine by reason of their occupations.

Goring concludes from a statistical study of the English convict that, "Offenders committing crimes of violence are on a whole rather less anæmic and have a ruddier complexion than criminals generally."

#### TATTOOING

The practice of tattooing is very common among prisoners, but it is more or less limited to the members of the lower strata of criminal society. The forger and the convicts of this type are not given to this mode of adornment, since it makes identification more easy and certain. Young criminals may in a spirit of bravado submit to this painful form of decoration to display their "nerve" and ability to endure pain.

I have found all portions of the body tattooed, even including the genital organs, but the most common location of these "cutaneous embellishments" is the inner surface of the forearm. All manner of figures are found, common among them are initials and names, religious symbols, crucifixes, nude female figures, dancing girls, ships, stars, geometrical markings, obscene legends, birds, insects, dragons and designs for good luck, such as horseshoes, etc.

One reason offered for this practice is that the tattoo marks prevent blood poison in the case of injury; another, that they bring good luck. Burglars recognizing the dangerous character of their hazardous occupation have been tattooed for means of identification should they meet a violent death.

## HEART

I have noticed an irregular cardiac rhythm in many prisoners at the time of entrance to prison. This condition is, no doubt, brought about by alcoholism, irregular hours of sleep, excitement and sexual excesses.

The regime of prison life has a quieting influence on this functional tachycardia, as the prisoner is compelled to live a regular life, free from excitement and dissipation of all sorts.

Diseases of the heart are very common among criminals as is shown by the entrance examinations and the hospital records at the Indiana State Prison. Every form of valvular trouble has been noted, but the most common lesions were those which are to be found associated with arteriosclerosis. It is a well-known fact that disease of this vital organ has among its symptoms states of irritability, and in criminals this condition is often given expression in an inclination to violence. Periods of depression are also common. The intimate relationship between the nervous and the vascular systems explains the variability of the emotions of the individual who suffers with a pronounced form of heart disease.

Ellis, in his text on the "Criminal," gives the following interesting account: "Out of fifty-four examined by Flesch, 20 per cent. died of heart disease; 50 per cent. showed affections of the heart. Valvular insufficiency and cardiac atrophy seem to be remarkably prevalent. Penta found endarteritis and atheroma in eighty-two of his one hundred and eighty-four instinctive criminals, i.e., 44 per cent., although many of them were young. The condition," he says, "was diffused and pronounced; twenty of these eighty-two showed aortic insufficiency. It may be noted that arterial anomalies are extremely frequent. Thus Guerra found fourteen arterial anomalies in his eighteen criminals as against four in his twelve normal persons."

My personal experience has led me to believe that syphilis was responsible for at least 20 per cent. of the heart lesions I found.

## LEFT-HANDEDNESS

It has been the common assertion that a large number of prisoners are left-handed, but I am of the opinion that this claim is somewhat

of an exaggeration, though possibly the percentage of left-handedness is slightly higher among convicts than among free individuals.

Goring, in speaking of this condition, declares: "Comparative statistics of left-handedness are remarkably few and for Englishmen practically non-existent. Among 266,270 German recruits 3.88 per cent. are left-handed, a proportion practically identical with our percentage of criminals with this peculiarity."

#### NERVOUS SENSIBILITY

I find that among a certain class of criminals, such as hysterics and malingerers, there is a tendency to exaggeration of the slightest pain, or any form of physical discomfort; this is especially noticeable among those who attempt to evade daily tasks and work assigned them. Indeed, the morning sick lines in penal institutions are full of prisoners pretending to be ill and furnishing only the most flimsy excuses for such claims. On the other hand, however, I have noticed an astonishing degree of sensory disturbances, especially those of anæsthesia and paræsthesia.

I have performed many minor surgical operations on convicts without general or local anæsthesia when no complaint was made of pain. In very severe injuries I have sometimes found it necessary to exercise authority under the threat of demerit to keep injured convicts in bed.

I have known prisoners to amputate their fingers and otherwise mutilate their bodies without apparently suffering pain. These self-inflicted mutilations were usually made for the purpose of avoiding labor. In one instance it required twenty-seven deep and superficial stitches to close a gaping wound, made with a sharp knife, across the shoulder and arm of a prisoner while in a fight with a fellow convict. The severed ends of the muscles were sewed together and the edges of the skin approximated without the slightest sign of pain on the part of the patient.

#### GENITALIA

I have observed that the genitalia of prisoners are usually well developed and the reason for this is very apparent, since most of them are given to very frequent sexual intercourse and masturbation.

"The sexual sense of the youthful male criminal is usually in-

tense and previous to apprehension was gratified physiologically unless it was perverse or exaggerated; in which case, onanism or pæderasty were resorted to."

The life in penal institutions does not seem to suppress or diminish the genetic desires, and since the opportunities for intercourse are lacking, inverse and perverse sexual habits are acquired in not a few instances. In those prisons where for lack of room it is necessary to put more than one prisoner in a cell, the practice of sodomy and other forms of homosexuality must be expected.

#### GENERAL CONSIDERATION OF ANTHROPOLOGICAL DATA

It is extremely gratifying, no doubt, to the disciples of Lombroso to have such a worthy opponent as Goring to declare: "The thief, who is caught thieving, has a smaller head and a narrower forehead than the man who arrests him; but this is the case not because, of the two, he is more markedly inferior in stature.

"The incendiary is more emotionally unstable, more lacking in control, more refractory in conduct, and more dirty in habits, etc., than the thief; and the thief is more distinguished by the above peculiarities than the forger; and all criminals display these qualities to a more marked extent than does the law-abiding public, not because any of these classes are more criminal than the other, but because of their inter-differentiation in general intelligence.

"From our statistical evidence, one assertion can be dogmatically made; it is, that the criminal is differentiated by inferior stature, by defective intelligence, and to some extent, by his anti-social proclivities.

"The following figures, however, may assist the imagination in realizing the nature and proportions of this differentiation. We may take it that one in thirteen persons of the general population are convicted at some time of life for indictable offenses. If the total adult population were made to file by in groups of thirteen, and out of each group one person was selected who happened to be one of the smallest there in stature, or the most defective in intelligence, or who possessed volitional anti-social proclivities to a more marked degree than his fellows in the group, the band of individuals resulting from this selection would—in physical, mental, and moral constitution—approximate more closely to our criminal population than the residue.

"We find, also, that crimes of violence are associated with the

finer development, with the more marked degrees of ungovernable temper, obstinacy of purpose and inebriety, and with the greater amount of insane and suicidal proclivity, of persons convicted of these offenses; and that tall persons are relatively immune from conviction of rape; and that fraudulent offenders are relatively free from the constitutional determinants which appear to conduce to other forms of crime."

If it were possible to measure physically and mentally one thousand prisoners and one thousand average free persons, I am convinced that the criminals as a class, allowing, of course, for individual differences, would show physical and mental stigmata with a greater regularity and in a more marked degree than would the equal number of non-criminal civilians.

Goring, who denominates the Lombrosian idea of crime as a superstition, and sets out to prove that it was, comes to the following conclusions, after the comparative study of four thousand English convicts and an equal number of university men:

1. The criminal is mentally and physically defective.
2. That environment bears but an insignificant causal relationship to crime.
3. That criminal traits and tendencies are inherited in the same manner as tuberculosis.
4. That classes of criminals may be differentiated one from another by physical and mental attributes.

While many efforts have been carefully, honestly, and scientifically made by anthropologists to determine whether or not there are corporal differences between those who have been convicted of crime and those who have not, there has not yet been furnished sufficient definite data to warrant positive conclusions.

We are therefore justified, especially in view of the findings of the positivistic school of criminologists, to conclude that there is no distinct criminal class. There are no signs or symptoms which so mark or label an individual that we may say with absolute certainty that he will or will not be a criminal, but we are compelled to acknowledge that the stamp of constitutional inferiority is indelibly impressed upon these individuals who constitute our prison populations.

Is it not possible in our zeal to obtain anthropometric data that we have lost sight of the efficient factors of crime? Has there not been

a tendency to overestimate the value of mere mathematics and measurements? We have jumbled statistics until we are practically lost in the maze of confusing and misleading arithmetical calculations.

We must ever bear in mind that the criminal is a human being, that the biological element is to be considered, the environment, the diseases or defects which govern, regulate, and modify the mental activities are of far more value than bodily measurements. The anthropological study of crime must give way to the methods of psychological investigations.

In very large proportions, the real, though not always the apparent, force for criminality was mental defect, and this generic term includes insanity, epilepsy, feeble-mindedness, borderline or psychopathic states, and with these states of mental aberration the criminologist has to deal.

No better conclusion or summary of this lecture can be made than by quoting the graphic description of criminals given by Dr. J. S. Wight, who says: "The concurrent and unanimous testimony of those who are, from their experience and knowledge, most competent to judge, is: That the under class of criminals have more or less defective organizations, especially as relates to their nervous system and more especially to their brain; that they are more or less deficient in moral sense, showing in this respect the lack of development or the result of decay, the best and last developed sense, the moral sense, disintegrating first of all; that they are perversely wicked and indomitably inexpedient, committing crimes when doing right would be of more use to them; that they are as passionate as the wild beasts of the forests, and as restless as the ocean that heaves at every gust of the wind; that they are at war with mankind and ever in commotion with themselves; that they are, like the ship beaten out by the storm—the ship without compass, rudder, or captain; that they are formed and fashioned by the hand of an evil genius, whose name is bad heredity, and whose hand-maid is ignorance; and that they cannot be very much reformed, and that their reformation ought to have been begun in their ancestors."



## A PSYCHOANALYTIC REPORT OF A CASE OF MUTUAL AMBIVALENT TRANSFERENCE

By L. D. HUBBARD, M.D.

IN his paper on the "Theory of Schizophrenic Negativism," Bleuler says: "When a normal person loves something or somebody on account of one quality, but hates them on account of another, the result is not an entirely unitary feeling tone, either the positive or negative outweighing at times." In our dealings with the insane it is very frequently noted that the hate side of this feeling for people which is successfully repressed up to the outbreak of the psychosis, is the only side which makes itself manifest during the mental illness. In the neuroses, too, this reversal of the usual state of feeling is often seen. No matter how completely the love element may seem to be replaced by the hate, that it has not been completely destroyed is proven by the unconscious effect it has on the life of the individual.

While working in a neurological clinic in a nearby city a case came to my attention which illustrated to an unusual degree the effect of family complexes and the ambivalent reaction to them upon the selection of friends. The circumstances came out in a partial analysis of a young woman who came to the dispensary for relief from numerous neurotic symptoms. She had a close friend with whom she lived and with whom she was making a very satisfactory social adjustment. The friendship had sprung up spontaneously on both sides on first acquaintance, but the factors which lay behind the choice of each woman remained unconscious until the analysis had been carried some distance, when the patient suddenly became aware of the basis of her attachment and proceeded to analyze with her friend the latter's attachment to her.

Miss A., the patient, was suffering from a repression neurosis dependent largely upon her struggle to keep unconscious a marked antagonism against her mother. She was an only child, two other children having died before her birth, and she had been hedged about on all sides by her mother's overzealous care and had felt herself more or less hampered since her adolescence by her mother's ever-increasing invalidism. She specially resented her mother's natural

interest in her affairs and her demonstrations of affection which she met with a scarcely repressed recoil. The mother likewise showed a good deal of sentiment at times and this was especially obnoxious to the patient. Not long after adolescence a reaction set in against all her early training. Brought up by educated parents and taught a wholesome respect for the English language, she now constantly used certain ungrammatical phrases and slang expressions which never failed to call forth a horrified rebuke from her mother. Her reading had been along classical lines as a child, but her revolt led her into the most frivolous of light literature and this also to her mother's distress. Furthermore, she developed an unreasonable reaction against religion and all things even remotely pertaining to it, and her scorn of her mother's religious professions was only thinly veiled.

Having left her home to work in a distant city, surrounded by strangers, she made no intimate friends for a year or more. She had many acquaintances amongst her fellow-workers, but not one with whom she shared her deeper feelings. Her adjustment to her environment was superficially normal, but she was constantly annoyed by one or another neurotic symptom. She wrote daily letters to her mother and sent frequent gifts which she could ill afford. Then there moved into the same boarding house another woman, Miss B., to whom the patient at once took a great liking. She became acquainted with her and immediately set about inventing excuses to call upon her or to walk to work with her. She made no attempt to analyze this attraction, but soon found a superficial mutual interest to which she laid the rapidly increasing friendship. Though it commenced as a rather impetuous emotional attachment, it settled down after a few weeks to a mutually satisfactory normal friendship to the marked benefit of both parties. While it was still in the emotionally unstable stage, Miss A. came to the dispensary for treatment. During the first few treatments a great deal came to light regarding the patient's attitude toward her mother, but it was some time before her antagonism was openly admitted. One day she stated that on the day before, when she had wished to call Miss B.'s attention to something, she had said, "Look, Mother." She was quite amused by this as she could see no way in which her friend resembled her mother, either in character or appearance. It was not long, however, before a strikingly large number of points of similarity became evident, once attention

was called to the matter. Miss B. was herself an excellent English scholar and was sometimes distressed by reactionary outrages against the language on the part of Miss A. This the patient did not resent, but other resemblances caused an outburst of emotion quite out of all proportion to the exciting cause. A mere suggestion on the part of Miss B. that she might go to church the following Sunday precipitated a torrent of tears, and it was several days before Miss A. could be convinced that such a suggestion did not necessarily blight their entire friendship. Some slight evidences of sentimentality shown by Miss B. provoked similar outbursts. Miss B.'s fondness for classic literature, music and art brought forth only rather caustic comment. Countless little modes of expression or minor habits reminded Miss A. of her mother and when noted brought a momentary disquietude. Despite these occasional upheavals, however, the friendship flourished and since its stabilization Miss A. has made a much healthier adjustment to life.

Miss B. was one of a large family, a brilliant scholar, but of physically inferior make-up. As a child she was dominated by her older sister and the greatest conflicts of her life revolved about her. The sister held this power largely through her power to say unkind things, to ridicule Miss B. and to impute to her unworthy motives. Though the situation was alleviated by the separation of the sisters and Miss B. regarded her with the sincerest love and admiration, the memory of the many conflicts remained at the time of her acquaintance with Miss A. In this case the unconscious association came to light through a dream in which Miss A. and the sister were interchangeable. Following this innumerable resemblances became evident. Miss A. had the same ability to vent her anger by making unkind remarks. In fact, she must have reacted to Miss B. with all the spite which she had suppressed from her mother. She had a certain rapidity of movement and impetuosity of temper very similar to the older sister and a thousand little traits reminded Miss B. of the old conflicts.

Here we have two women suppressing antagonism for female members of their families—antagonism so marked that any prolonged companionship is out of the question. Thrown into a new environment, surrounded by plenty of other women amongst whom they are at liberty to choose their friends, they are irresistibly attracted to one another,

though each in the end turns out to bear a close resemblance to the chief factor in the other's conflict. Miss A. reacts to those features of resemblance with unreasonable outbursts of emotion. Miss B. sees the resemblance but shows no abnormal reaction. Now Miss A. spontaneously states that those characteristics which attracted her to Miss B. were those which reminded her of her mother, quite forgetting that in the early part of the analysis she denied any such association. Taken out of the setting wherein those characteristics brought about antagonistic reaction they were devoid of harm and expressed themselves in that attachment which normally belonged to the mother. The love element of the ambivalent feeling, working unconsciously while the hate against the mother was strongest, brought into being the close friendship with that individual in the environment whom most closely resembled the mother.



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